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UNSOLVED PROBLEMS SUGGESTED BY CEREBELLAR CONNECTIONS AND CEREBELLAR HISTOLOGY *

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The neurologic sciences probably have never experienced the dissociation between the anatomic and physiologic points of view that is so bitterly complained of elsewhere in the present dynamic welter. Consequently, probably everyone who has been occupied with neuro-anatomic problems has always sought to interpret his observations more or less in terms of function as well. The purpose of this paper is to voice the obstacles of such functional interpretation which are due to lacunae in the knowledge of neuro-anatomy and of which nearly every one working in this line is aware; hence, the banal character of these few reflections—at least to neuro-anatomists. It may be that before this paper is published, some of the questions raised will have received their answers.

Broadly speaking, when one deals with the central nervous system as a whole or with any portion of its gray matter or even with any individual neuron, three questions arise: (*a*) What are the sources of the nerve fibers terminating in the structure in question, whether it be the whole central nervous system or certain masses of gray or single neurons, and what kind of nerve impulses do they convey to it? (*b*) What does the structure itself do to these impulses? (*c*) What is the distribution of the fibers emerging from it and what is the ultimate distribution and effect of the efferent impulses conveyed by such fibers?

Investigations of the nervous system, whether anatomic or physiologic, must always be an attack on some special portion of it or some particular function. This analytic approach has been criticized—both in neurology and in other branches of science. One hears such statements as that the body, or the nervous system, or the cortex, "functions as a whole." This statement may be true, but it is irrelevant as a protest against analysis as a necessary preliminary to synthesis. It is, however, a good corrective to any tendency to regard the nervous system as a congeries of circuits acting independently of one another. The importance of the various integrations before the lowest levels are

* Read at a meeting of the Association for Research in Nervous and Mental Disease, New York, December, 1926.

reached, illustrated in Tilney and Riley's book, is better realized than formerly. Voluntary movements, for example, may partially disappear with injury of the pyramidal tract, but no one considers this tract as the sole carrier of all impulses necessary for the normal performance of such voluntary movements. How far lower reflex circuits can act independently, how far they maintain their independence with the increasing development of forebrain and correlated mechanisms, and the nature of the influence of such higher centers on them have always been fundamental problems in neurology. It would appear, however, that modification of the activity of one circuit by another would be accomplished mainly by some form of combination with the afferent impulses received by the modified center from other sources. More and more, too, there emerges the importance of afferent impulses acting constantly and (consequently?) not occupying the field of attention, such as the protopathic, especially visceral, field whose dissociated activities have so occupied the attention of the psychoanalyst, and the proprioceptive—especially the tonic proprioceptive—field so germane to cerebellar discussion.

THE CEREBELLUM

Since Sherrington's work, it has been customary to speak of the cerebellum as the great proprioceptive "ganglion" or coordinating center. That this statement throws great light on the nature of the cerebellum is true, but—as will appear later—certain qualifications apparently must be made. This must not be taken to minimize the obvious but nevertheless important truth that one great ultimate source of all impulses regulating—not necessarily initiating—all movement as well as tonus and that distribution of tonus known as posture must be the stimuli arising in the organs of movement and posture, the muscles and tendons themselves. In all the diverse normal reactions of the voluntary musculature to various stimuli, however coordinated by the central nervous system, this proprioceptive element must be present.

The following applies principally to mammals and especially to man:

RECEPTORS

Starting with the peripheral sources of afferent impulses to the cerebellum; is this proprioceptive source established from the point of view of purely anatomic observations? It seems to me probable rather than absolutely proved. There are no degeneration experiments showing that neurons terminating peripherally in muscle-tendon spindles and perhaps other proprioceptors terminate centrally around Clarke's column. On the other hand, much evidence exists that many of the coarse fibers running up the dorsal column and giving offsets to Clarke's column originate from a certain type or types of larger spinal ganglion cells which in turn send their peripheral processes to proprioceptors. Along with these, in the dorsal column, however, are probably fibers

from the corpuscles of Pacini and Meissner, the latter probably carrying tactile impulses. There has at present, as far as I am aware, been no differentiation made between these categories as regards termination in Clarke's column—or indeed elsewhere. So, while it is highly probable that the fibers ending in Clarke's column are in part proprioceptive (position and movement), there is no strict anatomic proof that other kinds of impulses may not also be received. It may be remarked that I have never seen any basis for assigning to Clarke's column or to the cerebellum any visceral function. Also, while the term proprioceptive is useful, it has likewise led to confusion. The same applies to the term autonomic. I confess that I am uncertain as to the exact meaning of these terms in Winkler's great work. Would it not be better, in protocols of sensory examinations at least, to state simply the implement and modus operandi of the tests and not to use terms of various categories of sensibility?

It is obvious from the foregoing that there cannot be any proof as to the respective rôles played by the tendon and the muscle spindles. The interesting phenomenon—which I believe often occurs—that in a tenotomy—say of the Achilles tendon—the tendon reflex is lost but the sensation of movement and position is retained would seem to indicate that the tendon and articular receptors mediate the kinetic reflex and the muscle spindles the sensation. Further observations might be made on this point.

SPINAL AFFERENT CONNECTIONS

In considering the cord, one may note that, according to statements in some books, the fibers ending in Clarke's column are terminals of ascending fibers of medium length. According to Cajal's observations, the fibers entering this column are principally not terminals but are collateral branches. While it is important to verify in adult man many of the observations as to collaterals made by Golgi's method on young or embryo mammals other than man, this observation of Cajal's is in accord with the fact, easily seen in silver preparations of the adult human cord, that the fibers entering Clarke's column from the dorsal white column are of smaller caliber than the great majority of the fibers of the dorsal column. This being the case, it seems probable that at least some of the individual proprioceptive fibers entering the dorsal column may convey the stimuli traveling along them, not only to Clarke's column and thence to the cerebellum, but also to the nuclei of the dorsal columns and thence to the thalamus and cortex, to which should be added collaterals for cord reflexes and possibly other destinations. Such facts have an interesting bearing on the problems of timing, summation and also on certain questions concerning determination of paths by least resistance. It is also evident that tabetic ataxia, especially if due to a radiculitis, is not a pure cortical, i. e., sensory, ataxia, but

is of a combined cerebellar and cortical form, plus perhaps other factors. The general functional rôle of collaterals is one of the fundamental problems of neurophysiology. According to current views, the thin collaterals would conduct more slowly than the main fibers of which they are branches. They would also apparently conduct impulses of less intensity, or volume. Important general deductions might be made from these peculiarities, if true.

Another question that arises is the question of localization in Clarke's column. The radicular lamination of the longer ascending root arms in the dorsal column is well known and is in fact almost a corollary of the invariable relation of entering roots to dorsal gray matter. The collaterals from entering root fibers, exclusive of those to the zone of Lissauer, appear to follow a general law, necessitated, one might say, by the arrangement described. The collaterals given off first after entry naturally go through the gelatinosa into the plexus beyond, next those curving around its mesial side, and last, those to the most mesial gray of the cord, i. e., Clarke's column. One result of this is that, at any given level, the outer or lateral part of the column of Clarke receives collaterals from roots entering higher than those sending collaterals to its mesial part. That this is the case can be seen from a study of successive levels above a low cord crush. Just above the crush, the column of Clarke is destitute of entering dorsal column collaterals; higher, those collaterals from roots below begin to enter its lateral portion, and still higher the mesial portion is filled in.

Another perhaps more important result of the easily made observation that the column of Clarke receives collaterals from the middle part of the dorsal column is that these fibers come from segments below the level of the entry of their collaterals into Clarke's column. When this is coupled with the fact that Clarke's column is well developed—in fact, is large—in the twelfth thoracic segment, it is obvious that while the dorsal spinocerebellar tract perhaps principally conveys impulses from the trunk to the cerebellum it also conveys impulses from the lower extremity. It would be interesting to ascertain whether this bears any relation either to the segmental innervation or the segmentary innervation of the lower extremity. If one assumes that the fibers entering the lower part of Clarke's column are principally from the higher lumbar segments, one reaches the interesting conclusion that the cerebellum receives principally, as far as the lower extremity and the dorsal spinocerebellar tract is concerned, impulses from the flexors of the thigh and extensors of the leg. This conclusion, in view of the shortness of the lumbosacral segments, cannot be drawn with any degree of probability; *a priori*, in view of the trunkal character of the column of Clarke, one would expect the proximal segment—the thigh—to be favored. In questions of this kind, the descending arms of dorsal root fibers must also be taken into account.

Such considerations as the one described in the foregoing show how much is still to be done, especially on the human cord, in the finer analysis of its systems of fibers. There is still need of detailed study of secondary degeneration of the cord resulting from definite lesions in conjunction with clinical observations. As far as possible the result should be recorded by careful level photographs rather than by the prevailing simple line drawings so that the photographs can be used for other interpretations as well. It may be said in general that a comparison of figures in textbooks reveals a surprising amount of variation in the localization of the principal tracts, with little mention of level variations. A friend of mine once remarked that the surgeons are always looking for something to cut. This remark was made apropos of the section of the spinothalamic tract to abolish painful sensations from certain parts of the body. In how many of these priceless lesions have their scientific possibilities been fully exploited? In how many of them have there been comprehensive and thorough preoperative and postoperative sensory examinations? Are even clinical cases, in which operation or autopsy were not done, fully utilized? Some textbooks speak of a second pain path, for deep pain, in the dorsal columns. A case was presented at a certain conference recently of a patient with loss of superficial pain in one lower extremity and part of the trunk on that side, opposite signs of slight involvement of the pyramidal tract and no disturbance of the sense of position and movement. Pressure pain and pain on extreme flexion and extension at the joints (rarely tested, but important in such questions) were also absent, clearly showing the improbability of any such pathway of pain in the dorsal column.

From what has been said, the conception is gained of a segmental arrangement of Clarke's column, each root termination occupying a certain segment separated from adjoining segments by oblique boundaries. Is there any arrangement in the dorsal spinocerebellar tract? As is well known, Collier and Buzzard some time ago thought that there was a lamination of this tract, the lower segment being dorsolateral in the upper levels. This arrangement would not be in accordance with the general law of excentricity of the longest fibers, but the factor on which this law depends must be taken into consideration, i. e., that the last fibers to join a system naturally lie next to the gray matter of their origin, which, in a general way, is within the cord. The fibers, however, passing from Clarke's column to the tract in question in most cases take a directly lateral course to the ventral part of the dorsal spinocerebellar area. This would naturally result in a mainly dorsoventral lamination, the dorsal laminae in a higher level being composed of those fibers arising from the lower segments of Clarke's column. In sections below a cervical crush in which there was

"retrograde" or traumatic degeneration of the ascending tracts, the fibers of this tract persisting—at some distance below the lesion—occupied the dorsal part of the tract. As the fibers most apt to resist the trauma would be those from cells most remote from the lesion, this observation would tend to show a dorsoventral lamination. On the other hand, a lumbosacral crush showed a diffuse degeneration throughout the dorsal spinocerebellar area in thoracic sections. It may be that higher levels would have shown more evidence of lamination.

The ventral spinocerebellar tract, in regard to the location of its cells of origin, including their segmental locations, appears to differ from the dorsal tract. To some extent, at least, it appears to fill out the deficiencies of the dorsal tract, especially as far as the neck (and limbs?) is concerned.

As shown by MacNalty and Horsley and as pointed out also by Kappers, the projection of the spinocerebellar tracts on the cerebellar vermis does not agree well with the fore and aft localization of head, neck, limbs and trunk, there being a mixture of segmental representation. While this does not necessarily negative strict cerebellar localization entirely, it still is adverse to it. Such observations are of great importance in other respects as bearing on theories of cerebellar function and should be extended further. A careful study and comparison should be made of secondary degenerations following lesions at various levels of the human spinal cord. Such material can be collected, and it may be pointed out that while only a short period—from one to three weeks—after the incidence of the lesion is most favorable for Marchi preparations, the "Marchi bodies" remain a much longer period and would be available for work of this kind when studies of the finer fibers are not necessary. Accurate information is of importance here not only as bearing on localization but also as on it hinges the general interpretation of cerebellar activity.

BULBAR AFFERENT CONNECTIONS

When one turns to bulbar afferent connections, one finds even more important gaps in the present anatomic knowledge. Among the minor ones are the terminations in the cerebellum of the contingents from the nuclei of the lateral columns and from the external arcuate nuclei. Regarding the latter—and to some extent the former—ignorance also exists as to the possible nature of the impulses they convey. Another interesting question, not much emphasized, is the termination of collaterals from spinocerebellar fibers in the bulbar lateral column and the vestibular terminal nuclei and the giving off of collaterals from the olivocerebellar fibers. These points, some of which have been observed by Collier and Buzzard and others, have been stressed by Lorente de Nó, who emphasized the fact that all the various afferent

proprioceptive fibers to the cerebellum thus establish lower circuits, which may account for the compensation made possible thereby for the maintenance of the righting reflexes of Magnus-de Kleijn after destruction of the cerebellum. An important question is the connection of the nuclei of the dorsal columns with the cerebellum. The discrepancies here between observations on normal human brain stems and experimental work in other forms are well known. This may be explained possibly by Tilney's observations that the external or large-celled nucleus of von Monakow or Blumenau, or the external nucleus of the column of Burdach, may not be present in lower forms of life and may furnish such a connection in man. In connection with this it may be remarked that two apparently—but only apparently—antagonistic tendencies are emerging from comparative work, pioneered by C. L. Herrick and Edinger, and now being carried on more intensively, especially here and in the Netherlands, i. e.: (1) the great light thrown on the nature of the human central nervous system by such studies, and (2) the increasing revelation of great differences between the central nervous systems of man and the usual experimental animals and the consequent great need of caution in applying *in toto* to man the results—especially physiologic—obtained on such forms. Dr. Pike has always emphasized the great shifting of functional emphasis as one ascends to the anthropoids and man. There is great need for much experimental work, both physiologic and anatomic, on the anthropoids and still more utilization of human material. If only the craving of the general pathologist to carve the brain into unusable slices could be curbed or sublimated into a more careful macrotoming into more practical sections for future study, this end might be more easily attained.

It seems fairly clear that the paleocerebellum receives proprioceptive stimuli from the organs of somatic movement by way of spinocerebellar and probably by certain afferent cranial nerve secondary tracts and also from that proprioceptive organ of the head—the vestibule. Regarding the latter, there is again uncertainty both as to whether and to what degree the cerebellum receives direct root fibers or secondary tracts and whether the projection is on an internal nucleus, the nucleus fastigii, principally, or directly on the cortex, and the exact distribution of vestibular impulses on the cerebellar cortex. Allied unsettled points concern the variable striae medullares—now apparently not representing secondary cochlear tracts—together with the perpendicular fibers in the raphe, to which may be added those in the pons. Attention might be called here to the interesting but, to me, unsupported view of Winkler that Bechterew's nucleus is autonomic and also to the interesting Spanish observations showing the presence of efferent fibers in the cochlear root —but not from Bechterew's nucleus. There is also some uncertainty

as to what proportion of the fibers connecting the bulbar vestibular field with the cerebellum are afferent and efferent, respectively.

In general, respecting these afferent paleocerebellar connections, it may be remarked that while they are overshadowed in man by the more recent olivocerebellar and pontocerebellar connections to the neocerebellum, they are still well developed and are insignificant only relatively to the latter.

INFERIOR OLIVARY CONNECTIONS

It is when one comes to the inferior olive, however, that a deficiency in knowledge is revealed which, to my mind, bars the way to a clear anatomicophysiologic interpretation of the cerebellum. The distribution of the inferior olivary fibers on the human cerebellar cortex, thanks to the work of Holmes and Stewart, appears to be fairly well known, and it is important to note that they apparently terminate in the vermis as well as in the hemispheres, although restricted by some observers to portions of vermis and hemispheres. This connection is probably not as new as the pontile connection. It is also interesting to note a correspondence of certain parts of the olive with certain parts of the cerebellar cortex. This tendency to correspondence is marked in other connections such as the optic pathway and is especially striking in the topographic correspondence between the sensory paths from legs, arms and head in the bulb and in the cerebral hemispheres, the legs in both cases occupying the medial position. In the case of the sensory path, the correspondence appears to be one of the expressions of bilateral symmetry.

The great gap in the knowledge of the olivary connections is the ignorance of the source of the fibers ending in the olives. That the central tegmental tract and probably that of Helweg are the main, possibly only, afferent olivary connections is well established, but the exact source of their fibers is not known. It must be remembered, though, that among the secondary ascending tracts in the ventral and lateral cord, comprising fibers to the bulb, paleocerebellum, tegmentum, colliculi and thalamus, are probably fibers to the olive, according to observations of Collier and Buzzard. The connections described are old ones, and it would be interesting to know if such spino-olivary fibers terminate only in the paleo-olive. On the other hand, as will be mentioned later, the tract of Helweg may be a newer acquisition.

The olives supply the vast majority of the fibers of the restiform body in man; they form one of the great cerebellar connections; they are mainly new structures but are not as recent as the pontile connection, there being probably a paleo-olive in all vertebrates, at least in all with well developed cerebellums; while the olives supply the vermis, their great development and linear convoluted character must

be attributed in part at least to the necessity of covering in man the much larger hemispheres as well as the vermis. It is in connections of this kind that the necessity of concentrating on studies of the anthropoids and man is manifest. The comparatively insignificant olives of even such mammals as the cat do not lend themselves to their investigation by experimental methods. Whether the central tegmental tract is thalamic or tegmental in origin or both will probably be discussed by others, but there will be a great difference in interpretation according to what view is held. It may be remarked in passing that it seems as though there should be some means of projecting ocular proprioceptive, in conjunction with retinal, impulses on the cerebellum. As Magnus has shown, the optic righting reflexes increase in importance as the mammalian scale is ascended. On the other hand, if one accepts a partial thalamic origin for this tract, may it not represent the projection on the cerebellum of the impulses necessary for the cerebellar regulation of various emotional and instinctive reactions performed by the somatic musculature, as opposed to a pallial cerebellar control of the various voluntary muscular activities by the palliopontile system? It will be noted that this is contradictory to another speculative suggestion given below regarding the palliopontile systems.

If, as has been held by some, the olive receives fibers from the nuclei of the dorsal column, an ample spread from the whole body on the whole cerebellum might be provided for. This dorsal column-olivary connection should be crossed. In connection with this may be mentioned Tilney's conception that the olivocerebellar connection supplies the proprioceptive impulses for the cerebellar regulation of the neokinetic activities. This view appeals to me, and such a dorsal column-olivary connection would provide, perhaps, the more ample olivary connection which it would apparently require. If, on the other hand, the olivary connection should turn out to be one conveying impulses from such higher centers as the thalamus and the striatum, the whole neocerebellum would probably receive little or no direct proprioceptive stimuli. The importance of this question in cerebellar interpretation is obvious.

Does the topographic projection of the inferior olive on the cerebellar cortex mean a similar topographic projection of the stimuli carried to it by this route? Obviously this question cannot be answered without a much more accurate knowledge of the afferent olivary systems such as the central tegmental tract. It must be pointed out here that absence of histologic evidence of isolated components in a conduction path does not necessarily militate against localization of stimuli traveling along it. There is no histologic evidence of such isolation in the nuclei of the dorsal columns of the cord, yet in all probability stimuli traverse them which enable the pallial cortex to discriminate between two points about 2 mm. apart. Histology simply shows diffuse intermingled terminations

of the fibers of the dorsal column in these masses of gray. This difficulty is solved if one remembers that, on the law of chances, no two terminals would hit the same aggregate of secondary neurons; some would be the same, and many would not. There is no one-to-one conduction of neurons, merely successive partial mixtures in successive nuclei, but on the pallial cortex cutaneous point 1 must activate a somewhat different though probably overlapping aggregate of neurons from that activated by point 2. As is seen in the olivary path and especially in the visual path, there is some topographic representation, but this is the result of overlapping groups, in all probability. I owe this general conception partly to Casamajor, as resulting from a discussion of the question with him some time ago. The bearing of this conception on questions of cerebral localization is obvious.

In the case of perception of movement and position, however, spatial discrimination is less important, and what appears to be a varying intensity of stimulus seems to be the basis of the cortical discrimination. Here the tension of a whole muscle or muscles and the attached tendons involves stimulation of many muscle-tendon receptors, and the discrimination—cortical or otherwise—of the degree of tension and movement would apparently depend on the intensity of their stimulation. As this apparently conflicts with the all-or-nothing principle, one must apparently, if one accepts the latter centrally, resort to postulating the stimulation of an increasing number of receptors with increasing tension. This can be done much in the same way that the upholders of this theory account for varying degrees of tone and movement by the stimulation of varying numbers of muscle fibers by motor nerve fibers. These considerations would apply equally well to the nonsensory proprioceptive projection on the cerebellum.

PONTILE CONNECTIONS

When one turns to that still more recent and still more massive afferent cerebellar connection in man—the palliopontile or middle peduncular one—it is with a more definite knowledge of the whole path, in this case from cortex cerebri to cortex cerebelli, but with important hiatuses. The first question is the important one as to whether the pontile fibers are distributed to the whole cerebellar cortex or whether all or part of the paleocerebellum (parts of vermis and flocculus) is exempted. There appears to be some difference of opinion here, and it may be remarked again that this anatomic question should be determined in man, with his large pallial connection, as well as in lower forms. *A priori,* it would seem more probable that certain pallial impulses, especially those for standing, would be integrated with the afferent ones from the body by the paleocerebellar cortex but, as will be seen later, this integration may be performed elsewhere.

When one examines the pontile nuclei, one finds that not so much is known as to topographic representation in the cerebellar cortex as in the case of the inferior olive—not even in respect to the two great divisions of palliopontile fibers—frontal and temporoparietal—terminating in them. However, there is some evidence (Masuda) that there is such topographic representation and that the pontocerebellar continuation of the frontal tract is projected on the caudal portion of the opposite cerebellar hemisphere and the temporal portion is projected on the cephalic. How can one get an anatomic clue as to the respective functions of these two divisions until their respective projections on the cerebellar cortex have been determined? In general, the pontile nuclei would appear to have equal claim to topographic localization as the olive, and yet it is not a laminar convoluted but a lump nucleus—an argument against the ultimate topographic character of the olive, perhaps. The difference may be simply a matter of space accommodation.

While the general cortical origins of the palliopontile tracts are probably known, it appears that vague ideas exist as to their exact meaning. A careful study of the much neglected—by neurologists—normal infant might be helpful, combined with a study of myelinogenesis. The order of development of higher centers concerned with movement apparently is, first, the older subcortical extrapyramidal systems, then the pyramids, and lastly the palliopontile systems. This seems roughly to coincide with the order of events to be observed in the infant: first, the athetoid and other normal involuntary movements and then the definite attempts (pyramidal) to do something. The latter are poorly coordinated and uncertain, the uncertainty probably being due not only to imperfectly discriminated sensory impressions but also to the imperfect action of the palliocerebellar mechanism. Incidentally, the normal infantile spastic grip, similar to that usually attributed to pyramidal lesions, may be mentioned, and a further study of the postures of infants, both when asleep and awake, recommended. And what rôle does the earlier developing paleocerebellar, as distinguished from the neocerebellar, mechanism play at this stage of development? The defects of coordination, including standing, seen in certain defective children, probably fall partly under this category. Granting, as seems to be the case, that the palliopontile projection is principally on the neocerebellum, may it not be that these connections exist principally to secure cerebellar cooperation in the more purely pallial, acquired, voluntary, pyramidal motor activities? Or should one say the more individualized and unilateral activities performed by the more recently acquired and increasingly important extremities, especially the hand? The view has been advanced that the temporoparietal projection is

especially in relation to a vestibular projection on the cortex by a presumably vestibulocollicular path. In this case, the frontal might be assumed to be in relation to the great somatic muscle-tendon proprioceptive projection, though not in close spatial relations with it. The question of rubral connections should be considered in relation to this, as will appear later. Pushing this line of thought further, may not another different distinction be made between the temporoparietal and frontal components? The relation of the former to possible vestibular centers has been emphasized by others, but might not this be extended to include a relation to all the surrounding sensory and gnostic areas? On the other hand, the frontal would have a relation perhaps to higher, more general and more synthesized motor manifestations, including also emotional expression especially. The latter might in this case bear the same relation to the palliothalamic outflow that the temporoparietal does to the pyramidal. However, the fact that there is some evidence that the frontal area has to do with standing—which would be in agreement with the frontal projection on caudal cerebellar hemispheres mentioned above—does not fit in entirely with this view. Standing and the proper maintenance of the center of gravity in progression and other activities are enormously complicated, especially when an upright posture is simultaneously maintained, and require a wide coordination of muscular adjustments—tonic as well as kinetic. The real antithesis of this would be the regulation of individual movements not so directly concerned in the latter maintenance. Further suggestions might be made, but what does one really know?

With the greatly increased interest in the subpallial extrapyramidal mechanisms, there should be renewed study of the pallial ones. Without a clear comprehension of their activities, there can be no complete grasp of the nature of pallial cortical activities in general.

Another obscure point which may be briefly mentioned is the existence of afferent rubrocerebellar fibers in the superior peduncle.

When one looks on the afferent cerebellar connections broadly, while there is no definite evidence of precise topographic localization, the distinction formulated by Clarke and Horsley seems to hold good: that, in general, the middle older portions of the cerebellum receive the more direct proprioceptive impulses from the body and head, especially from the older parts, i. e., the trunk and head, and that there is also a distinction between the vestibular and other bodily areas, while the lateral newer portions receive more of the olivary and especially of the pontile impulses. In spite of this, while it appears to be true that injury to any of these connections causes the disturbances of movement and posture ordinarily known as cerebellar ataxia, it is doubtful whether, at present, the character of the disturbance due to each can be clinically differentiated. When one considers the massive character of

the palliopontocerebellar and olivocerebellar connections, this inability seems almost pathetic.

It may be remarked, however, that clinical cases do appear to show a greater involvement of axial muscular mechanisms, or perhaps one should say of phylogenetically older bilateral movements, in lesions of the vermis.

It must be kept in mind, however, as will be shown later, that it is possible that there are no marked differences clinically between the effects of lesions of the various afferent systems.

EFFERENT CONNECTIONS

Juxtarestiform.—Before discussing the cerebellar cortex, if one looks at the efferent cerebellar connections, one again finds gaps which prevent a clear anatomicophysiologic comprehension of cerebellar activity.

That there are two principal outflows—one from the dentate, globose and emboliform nuclei via the superior penduncle, and the other from the nucleus fastigii via the juxtarestiform and uncinate fibers and thence, probably, principally by the various vestibulospinal and vestibulomesencephalic tracts—seems fairly clear.

Regarding the latter connection, it is interesting to note that, judging especially by the work of Clarke and Horsley, it drains more than the vestibular cortex and possibly more than the spinocerebellar areas as well. The question as to how far vestibular impulses are projected on the cortex arises here. If they stop at the nucleus fastigii, one finds an interesting arrangement, i. e., cortical integrations of another character acting on this primarily vestibular nucleus. In any case, the cerebellum appears in part to function as a higher circuit superimposed on lower vestibular reflex circuits for carrying other than vestibular integrations. The nature of this outlet would seem to point clearly to its being primarily for finer cerebellar vestibular integrations, using a common path with lower vestibular reflexes, but at present, and possibly from the earliest period, apparently carrying out cerebellar integrations of other than vestibular material. If an early cerebellar control of purely lateral line and vestibular material were established, it might throw light on the general nature of cerebellar control. These lower vestibular systems, vestibulospinal and vestibulomesencephalic, obviously can reach about all the voluntary musculatures. These paths are in the main simpler and more direct than the superior peduncular, but there are, nevertheless, probably connections with the noncerebellar extrapyramidal systems via, for example, the interstitial nucleus and the nucleus of the posterior commissure.

Speaking of the vestibulospinal tracts, who has ever used vestibular tests such as past-pointing, or pass-pointing, as an aid in diagnosing ventrolateral and ventral lesions of the cord? The vestibulospinal tracts

lying in this region of the cord undoubtedly carry down the vestibular impulses which cause deviation in such tests, and the deviations should be absent in lesions of the cord in the location described.

The general nature of the efferent paths of the lower vestibular circuits appears to be what one would expect in making the curious gyroscopic, plane-maintaining adjustments of limbs and eyes to translations of the whole animal through space. However, it is not improbable that the change in posture in man, as well as the increased control by the higher centers, may have thrown the adjustment out of gear or made it less important, except as a mechanism producing vertigo and nausea. Even these lower anatomic arrangements, however, have not been made entirely clear—much less the exact relation with and the influence on them of the superimposed cerebellar mechanism. It would be well, then, if one knew clinically the exact difference between disturbances due to lesions and stimulation along the more direct vestibular routes and those seen in cerebellar lesions. Holmes has shown differences in nystagmic movements, apparently. The view that the past-pointing reaction passes solely through the cerebellum is, I take it, not now so generally held as formerly, but it seems that there should be some alteration of this reaction in lesions of the superimposed cerebellar circuit.

Superior Peduncular.—Turning to the other great outlet, the superior peduncle, one is struck with the fact that, first a lineally extended convoluted nucleus, the nucleus dentatus, is encountered, and next a round massive nucleus, the nucleus ruber. The character of the former is due to the necessity of accommodating a great mass of cerebellar cortical fibers from the cells of Purkinje, which end in it. This is also shown by its absence in forms with a less developed cerebellar cortex. While there is still the possibility that some Purkinje axons emerge from the cerebellum without interruption, it does not seem probable that the number is considerable in the cerebellum of higher forms of life. It is the same necessity, in reverse, as that which produced the convoluted inferior olive, though in the case of the dentate there is concentration instead of diffusion; just as backward along the olive only tracts of obscure origin are encountered, so when one passes beyond the dentate only a massive nucleus and other obscure connections are encountered. In the case of the dentate, there is probably also an areal correspondence with the cortex; but what position does the nucleus ruber, its most important connection in the other direction occupy? Everything points to the latter as a great nodal point with many afferent and efferent connections both upward and downward and to its being used to carry the successively concentrated cerebellar influence in many directions, as well as to its being to some extent an important complex reflex center.

Incidentally, it may be remarked that the curious double-crossing of the dentatorubrospinal path is easily accounted for by the fact that the nucleus ruber is in connection, on the one hand, with higher centers exerting contralateral body control and with the cerebellum, which exerts an ipsilateral control.

Before discussing this further, it may be well to point out some crucial defects in the knowledge of this region and especially of the nucleus ruber—aside from the mystery involved in the fact that the extrapyramidal tangle is encountered.

One problem that arises is: Does the ruber receive paleocerebellar (i. e., spinocerebellar and vestibulocerebellar) influences? It seems probable that the globose and emboliform nuclei represent such a connection, but this, perhaps, is not established. If one assumes this to be true, do these nuclei innervate the paleoruber or the neoruber, or both? *A priori* morphologic considerations would favor the first view, but has it been established?

Again, is there a complete blending of efferent cerebellar influences in such structures as the dentate and rubral nuclei? Previous consideration of the nuclei of the dorsal column would seem to show that this cannot be assumed with certainty; but descending integrating systems are now being dealt with, and it is not for nothing that the pyramidal fibers, with their well marked topographic or segmental control, avoid interruptions in gray matter until they reach the level at which they act. If this "mixture" takes place, the synthesis of the whole or of large portions of the cerebellar cortex are projected on each of the peripheral motor mechanisms under cerebellar regulation, there being, however, considerable probability that the two outflows do differ physiologically from each other. Obviously, a more minute analysis of the structure described, if possible, is necessary in order to give an anatomic answer to this question.

One of the first facts that attracts attention is the apparently slight character of what seems to be the path of direct cerebellar control to the body by way of the superior peduncle. It is well agreed that the only well known path is via the rubrospinal tract. It is also agreed that this tract is comparatively small in man and that the cells from which it originates—the large paleorubral cells—are few in number. There are, perhaps, four explanations: one is that cells of the neoruber act as intercalated cells and that the impulses passing along the increased number of peduncular fibers are thereby further concentrated on the paleoruber. Even so, one would expect an increase in the old rubrospinal tract to meet the increased load, and such does not appear to be the case.

Another explanation is that there may be undiscovered descending tracts which perhaps have eluded observation because they are com-

posed of fine, possibly amyelinated, fibers. Here it may be remarked that while the absence of myelin probably does not have the same significance in all cases, yet amyelinated fibers are of a lower grade of development and it is not improbable that they may also, naturally, in some cases represent more recent acquisitions of the nervous system. The pyramidal tract as seen in some other mammals is a case in point, and the tract of Helweg may be another example. The discovery of other important descending rubral connections would simplify a comprehension of cerebellar activities.

A third explanation would emphasize the importance of those superior peduncular fibers which end in the reticular formation—aside from those to the oculomotor nuclei—and also the rubroreticular connection observed by von Monakow. This would give a polysynaptic descending reticulospinal path. Apropos of this, it may be remarked that degenerations reveal a large number of spinospinal fibers in the human cord, and it may be that this complicated, diffuse, spinal mechanism, no longer so much an independent spinal reflex apparatus in man, may be played on by descending reticulospinal and other descending extrapyramidal systems. This might account in part for the more diffuse character of many of the abnormal involuntary movements in man, as contrasted with the more individualized pyramidal movements, though the hyperkinesis or "mass reflex" seen in certain cases of transverse lesions of the cord is not, of course, identical with the abnormal movements "released" by higher lesions. Both the reticulospinal and the spinospinal systems might receive more consideration, but it seems doubtful if the reticular formation, after the known newer connections such as the neoruber and the central tegmental tract embedded in it, were subtracted, shows any greater development in man.

Still another explanation of the paucity of direct descending paths representing continuations of the efferent superior peduncular connection is that the newer path of the ruber represents connections with higher ne-encephalic parts of the brain. Present evidence favors this view, i. e., important connections with the pallium and neothalamus.

When the nature of the pallial connections with the ruber is considered, there appear to be three possible views: that the connection is rubropallial, palliorubral or both (von Monakow). There is perhaps an increasing tendency to regard the connection as principally rubropallial. The neothalamic connections may also represent part of a rubropallial path or may be part of a rubrothalamostriatal path. The latter, coupled with the existence of pallidorubral fibers, would give a return circuit to the ruber, and if one thinks that this necessitates a descending path for the resulting integrations, one is thrown back on the inadequate rubrospinal system. It must be remembered that the ruber probably has connections with the colliculi, which enable one to utilize the posterior or

medial longitudinal and predorsal bundles. On the other hand, reciprocal connections between two masses of gray matter may have a general physiologic function by modifying the intensity (reinforcing? inhibiting?), changing the timing or securing a more even diffusion by means of this back-lash. Such a general function would seem to be more likely, perhaps, when the reciprocal relation is between the same two structures, which would be the case, apparently, with the rubropallial connections but not with the rubrostriatal. Reciprocal connections are apparently present in many parts of the nervous system.

Pursuing the matter further, if the rubropallial character of much of the superior peduncular outflow, coupled with the existence of the palliopontocerebellar connection is accepted, one reaches the surprising conclusion that the palliocerebellar connections as a whole in the main represent reciprocal influence. Moreover, if the more direct cerebellar connections to the lower centers are considered as inadequate, one is driven to the conclusion that the main efferent path or paths for the projection of this vast reciprocal cerebellopallial integration on lower centers must be either by the pyramidal or the palliothalamic paths or by both; if by the palliothalamic path, it would probably ultimately impringe on the inadequate but only imperfectly known lower extrapyramidal paths, such as the rubrospinal and medial fasciculi; if by the pyramidal path, one reaches the interesting conclusion that the pyramidal tract has in it an indirect cerebellar component. This would explain the impossibility of voluntarily suppressing a cerebellar dysmetria—assuming such an impossibility exists—the only remedy being to subsitute a voluntary compensatory act calculated to reduce the disturbance; e. g., the dysmetric patient slows up or stops before a certain desired point is reached. A careful study of volitional movements in cases of cerebellar ataxia might disprove, but not prove this view. The study would not prove it because the pallial cerebellar components in normal voluntary movement, if transmitted via the palliopontocerebellubrospinal path, may be equally involuntary in character. It will be noted, parenthetically, that the pallial areas connected with the ruber are probably not, in the main, the same as those originating the palliopontile fibers.

If one reverts to the view that the connection between ruber and pallium is palliorubral, an additional load is placed on the rubrospinal tract.

This all shows the importance of a more definite knowledge regarding the size of this tract in man. Older investigators of the nervous system did much fiber counting, and there have been sporadic renewals of this more recently. Such investigations have more than an academic value as satisfying a fastidious demand for meticulous accuracy. Knowledge of the number of fibers in various fiber systems is one of

the foundation stones for a correct understanding of how the nervous system does its work.

When one passes beyond the fastigial and dentate nuclei, one is no longer dealing with purely intracerebellar integrations but with integrations of cerebellar influences with extracerebellar ones. In all these successive integrations, one must look to further advances in the general conceptions of interneuronal interaction combined with an even more accurate knowledge of finer histologic relations. The great mass of information placed in the hands of neurologists by Cajal has not even now been fully utilized and must also be amplified in many directions, including verification of certain details, especially concerning collaterals, in adult human and anthropoid material. In addition to this, all of the observations on normal myelin stained by the Weigert method and on degenerating myelin stained by the Marchi method must be verified in axis cylinder pictures.

When the efferent cerebellar connections described in the foregoing and also the probable rubrothalamostriatal path are viewed, it can at least be seen that the anatomic mechanism is present for what is also seen in clinical cases, i. e., that the cerebellar regulative activity is exerted on—or is present in—all activities of voluntary muscles from reflexes to complicated voluntary activities, even in those concerned in such highly skilled acts involving nonposture maintaining muscles as speech. It is also seen that, of the two great outflows, the one via the fastigium would apparently have more to do with the posture-maintaining mechanisms of a vestibular and spinal character, while the dentatogloboso-emboliform one would have more to do with all the reactions of the forebrain, both the learned voluntary ones and the more automatic, emotional and semivoluntary ones which fill out the reactions of the forebrain and which include postural adjustments as well as individual movements. This view, however, is based on the assumption of no complete or extensive overlap in the cerebellar areas drained by these two outlets. There is probably considerable overlap, at least in the drainage by the various internal cerebellar nuclei.

Symptomatology.—One question, referred to already by Walshe, is why the symptoms observed in cerebellar lesions—cerebellar ataxia—are not identical with those observed in lesions of the superior peduncle which, on the contrary, appear to be involuntary movements of a choreiform or choreoathetoid character. It must be remembered that in the case of superior peduncular lesions—on which more observations are needed, by the way—not all of the efferent cerebellar paths are cut; there still remains the fastigiovestibular outlet.

If one had at his disposal careful comparisons of the disorders due to lesions of each of these—as well as of each of the afferent connections—whether they can be differentiated or not, light would be thrown

on the nature of the cerebellar control. Another point that arises in connection with the discrepancy between the effects of cerebellar and superior peduncular lesions is: What is the difference between the ataxic and the abnormal involuntary movements alluded to in the foregoing? I take it that one, at least, of the important differences is that the latter are apparently more spontaneous. What this spontaneity probably means is that the latter are more easily elicited by afferent tonic stimuli acting while the part affected is apparently in complete rest, while it actually is moving little or none but is incompletely supported—in other words a species of static ataxia. These movements also are elicited or increased by effort and by emotion, and attention often has an effect on them—not always of the same character. That there is a cerebellar component in them follows from what has been said.

On the other hand, if the disturbances due to rubral lesions in man, in the cases cited by Rademaker, are considered, one finds that there is inability to assume, or ataxia, tremor, hypertonia and, occasionally, choreiform or athetoid movements occur in assuming and maintaining, an upright posture. In regard to the first two, at least, the picture closely corresponds to that seen in cerebellar lesions. In individual cases, the correspondence is close. In other words, one sees what might be expected in view of the massive cerebellorubral connection. The question of hypertonia will be mentioned later. There immediately arises, however, the questions raised by the apparently negative results (Magnus) of cerebellar extirpation on the rubral righting reflex center. It seems to me that there is no real difficulty here. When the animal was decerebrated, all pallial activities ceased. It has already been seen that the superior peduncle represents largely a continuation of the palliocerebellar path and consequently the cerebellar regulation of pallial activities. Broadly speaking, these activities in man are the result of vast multisensory mnemonic associations, probably represented largely by the "spontaneous" movements absent in the decerebrated experimental animals, and when they are absent their cerebellar regulation is also naturally absent. On the other hand, cerebellar injuries in otherwise intact animals do result in disturbance of movement and maintenance of posture. The foregoing results, then, are partly confirmatory of the view that the neocerebellum is in general a mechanism regulating pallial activities. It is extremely interesting, if Rademaker's views as to the function of the ruber are correct, to see that there is provided a complex reflex center, on which pallial activities can play in securing proper adjustments of the lower postural mechanisms during its activity. The increase of the ruber in man tends to confirm this view. How far there is a shift to the neoruber and what that shift consists of, is a further interesting question. One cannot admit that man can assume and maintain his normal upright posture without his cerebral hemispheres,

whatever the tonus distribution may be in their absence. In fact, the rigidity observed in Edinger's case of a decerebrate child cannot be attributed to a rubral lesion if the only rubral change was a secondary degeneration or atrophy of its ne-encephalic portion. This suprareflex regulative activity of the cerebellum is seen also by the experiments of Magnus and de Kleijn, which show that the various body, neck and vestibular tonic reflexes are intact when the cerebellum is removed. It may be remarked that the superior peduncle is the most massive afferent connection of the ruber and that in cases of unilateral neocerebellar agenesis the opposite ruber is practically absent—the neoruber, at least. In view of this, any experimental work interpreted as negative with respect to cerebellorubral influence must be regarded as either inadequate as to method or erroneous as to interpretation. Here anatomy must take precedence over experiment, if there is a conflict between the two.

To explain any discrepancy between the effects of cerebellar and superior peduncular lesions, one would, if he were strictly logical, first minimize the differences between the two kinds of disturbance of movement, and what differences there are would be attributed to the spinovestibulocerebellofastigiovestibulospinal mechanism being affected in the cerebellar but not in the superior peduncular lesion, or not so much so. It is logical that the activity of this split-off portion acting on lower centers, in part at least, accounts for the unequal tonic maintenance possibly underlying the disturbances seen in superior peduncular lesions. While anatomists cannot give the anatomic side of many functional phenomena, it is also true that the physiologist might follow—and is now following, by the way—more closely anatomic suggestions rather than the simple transverse cut, for example, in his experimental work; the clinician likewise can often follow the same suggestions in his observations. To take examples already known, the anatomist would not expect *a priori* to find sensory changes in cerebellar ataxia nor even in certain abnormal involuntary movements. Might not more be looked for, anatomic clues being used? Again, could not more be gained even without autopsy in all cases by carefully noting combinations and dissociations of clinical symptoms as Head did in his work on the central paths of sensation?

The discussion of extrapyramidal questions is to be avoided here for the double reason of their intricacy and difficulty and of my ignorance, but it may be remarked that many postures are not maintained by gravity tensions. Probably, nearly every one holds the fingers partly flexed, but not against gravity. Is this flexion due to greater strength of the flexors of the fingers, and they are "stronger" than the extensors; is it due to some mechanical arrangement of ligaments and bones, or is there a real slight difference of tone between the two groups? That there is such a neural "holding" in certain postures, in the case of

individual parts, possibly by spinal circuits, is shown by certain experiments. Just as the general bodily feeling of welfare or illness is conceived of as due, in part at least, to a constant stream of affective stimuli from viscera and environment, so must posture be conceived of as maintained, in considerable part at least, by a continuous stream of proprioceptive impulses from the organs of motion and holding. This stream splits, after entering the central nervous system, into many streams: to cord circuits, cerebellum, midbrain, basal ganglia and pallium. Probably all true ataxia and much hypotonia are due to cutting off one or more of these afferent streams to a given central structure or structures, or to injury of proprioceptive coordinating centers. It almost seems, on the other hand, that one could further generalize and say that abnormal involuntary movements and hypertonias are due to lesions more on the descending side. A lesion of the proprioceptive mechanisms must always result in disturbances of tone and movement.

While much of tonus is thus maintained, one cannot, perhaps in the broadest sense, conceive of all tonus or posture, or perhaps one should say continuously maintained muscular tensions, as due solely to proprioceptive stimuli. While tonus probably cannot be properly maintained pyramidally (i. e., by continuous voluntary effort), extrapyramidally, or reflexly, without the assistance of proprioceptive stimuli, into the tensions maintained in emotional states, mostly unconsciously or involuntarily, there also enter stimuli from different sources. These sources are probably affective, especially visceral, and also are endocrine. It may be that the latter influences are especially responsible for the tonic changes often attributed to lesions of the basal ganglia. This would be the component that would be most likely to have a peripheral sympathetic outflow if, as seems unlikely at present, a sympathetic tonic or kinetic influence on voluntary musculatures should be established. In fact, one may look broadly on the organism as a whole in a state of continuously maintained though changing endocrine, visceral and proprioceptive equilibrium repeatedly upset by clonic reactions induced principally, but by no means entirely, by exteroceptive stimuli, i. e., by more abrupt changes in these various internal and external stimuli. The cortex pallii receives its more immediate activation mainly, perhaps, from the last, but it blends the others as well.

PALLIOPONTOCEREBELLAR CONNECTIONS AND PYRAMID

In any case, in regard to the palliopontocerebellar connection, whether it is regarded as representing part of a reciprocal interaction between pallium and cerebellum with other pallial outlets as a final common path, or whether it is regarded as an indirect outlet for pallial influences via the rubrospinal and other tegmental paths, it is evident that the cerebellum receives impulses which are at the most only indirectly

proprioceptive. One may conceive of the pyramid as being set in activity by a synthesis of some sensory impulse, say exteroceptive (the immediate stimulus), linked mnemonically with a similar past stimulus ("gnosis"), with various past and present visceral and other protopathic impulses (the emotional, wish element), with past proprioceptive stimuli (skill or "praxis" and "conation"), and guided in its execution by concurrent proprioceptive stimuli. Accompanying this is a setting into activity of all the noncerebellar supplementary extrapyramidal machinery, with its tonic, sthenic and expressional contributions, and of the cerebellar mechanism with its regulation of the execution of pyramidal and extrapyramidal activities if one accepts the more usual view of a separate cerebellar outlet to the lower motor centers for the accessory palliocerebellar component. If the pyramid is regarded as the principal final outlet, the question (usually ignored) of the existence in adult man of pyramidal collaterals to the basal ganglia and the pontile nuclei as well as to the reticular formation, observed by Cajal, assumes even greater importance. Whichever view is taken, the palliocerebellar path would carry pallial syntheses to the cerebellum into which not improbably other than proprioceptive elements enter, and at the most such a proprioceptive influence would only reach the cerebellum by a circuitous route. In any case, the interesting fact emerges that all activities of voluntary musculatures must receive impulses that pass through the cerebellar cortex. If one regards the more primitive paleocerebellum in lower forms as coordinating proprioceptive stimuli and then handing them on to the lower motor and also to the more primitive motor centers of the forebrain, it is evident that more complicated relations come in with the pallium, when the cerebellum cannot be regarded as simply acting directly on motor mechanisms, whether there are important cerebellopallial connections or not. Perhaps further investigations of cephalad cerebellar connections in lower forms might throw more light on the nature of this mechanism.

Regarding the hyperactivity of certain reactions when "released" by interruption of connection with some other perhaps "higher" center, I sympathize with Pike's conception of this phenomenon, in line with Hughlings Jackson's view, that it is due more to an increase of impulses acting through the lower circuit than to a loss of some constant inhibitory influence exerted by the higher center on the latter. Stimuli which normally pass in part down through the pyramidal tract in lesions of it are diverted, so to speak, through the lower spastic-producing reflex circuit, on the basis of the ordinary view that the spasticity in question is due to a lesion of palliospinal fibers. It is true that one must think of the pyramidal influence in normal conditions as prevailing over other tonus-maintaining mechanisms; this must be the case for the pyramid to function, but the foregoing view emphasizes less some mystical continuous

restraining pyramidal influence than a more intelligible active stimulation. Regarding the question raised by some neurologists as to whether the spasticity usually attributed to a pyramidal lesion is really due in part or whole to a lesion of this tract, attention might be called to the importance of careful clinical observation in cases of inferior (hypoglossal) alternating hemiplegia, especially when unaccompanied by any disturbance of the sense of position and movement. In cases of this kind there is a pure pyramidal lesion without any involvement of extrapyramidal, afferent cerebellar or afferent pallial systems which might enter into questions of tonus, unless such fibers are present at this level in the pyramid itself. I have always supposed that there is no especial difference between the pyramidal signs in a lesion here than in lesions involving it at other levels.

The fact that the palliocerebellar path is open in cases of pyramidal lesions would seem to lead to the conclusion that the spasticity usually attributed to such lesions might be due, in part, to an overaction of the palliocerebellar system (if one accepts the view that the latter has a separate lower outlet) due to an increase of pallial cortical impulses acting on it in the absence of the pyramidal outlet or to an absence of an inhibiting influence normally exercised over it by the motor cortical areas. The spasticity and posture in pyramidal lesions do not appear to be what would arise from released, low segmental tonic circuits, but here again the neglected pyramidal collaterals must be taken into account. The mechanism of voluntary relaxation—which may be different from that of voluntary movement—should also be studied. Another possible explanation of the spasticity in question, if it is due to a pyramidal lesion, which should be taken more seriously into consideration, is that it is due to an overactivity of the palliothalamic or the palliorubral systems. The former, however, would more probably produce disturbances more of a choreo-athetoid character, which resemble distorted emotional expressions. The latter, the palliorubral system, should obviously demand more serious consideration.

CEREBELLAR CORTEX

From the preceding discussion, one possible view of cerebellar activity which emerges would show the cerebellum as a nodal point in which all its afferent impulses are mixed in a common pool and then distributed to various points, where they are successively integrated with other impulses until the final common path is reached. A partial view of this kind would concede a certain amount of "somatotopic" localization and a certain amount of difference between the activities of the paleocerebellum and the neocerebellum.

Would such a mechanism require a cortex? Would not a large lump nucleus with shorter axon neurons intercalated be adequate? The fact that the cortex is elaborate suggests a more complicated function.

It is strange that with the accurate knowledge of the cerebellar cortex it cannot be interpreted better, but, from what precedes, it is obvious that a great part of this difficulty arises from defects in the knowledge of the afferent and efferent cerebellar connections and their exact representation in the cerebellar cortex, as well as the ignorance of the general meaning of the various finer interneuronal connections.

The cerebellar cortex differs from the pallial in that it probably receives solely or mainly one kind of stimulus, direct protopathic and exteroceptive impulses probably being absent, and that there is no definite evidence of great mnemonic capacity. If the latter statement is true, it, considered with the late development of the cerebellum, would appear to show that the mnemonic activity of the pallial cortex does not rest on its late ontogenetic development—its immaturity at and after birth. Even here, however, it is thinkable, but not at all probable, that the skilled acts and also the various habits of posture and activity which make up the external manifestations of personality and which are probably represented by certain acquired arrangements of the inherited pallial cortex may be linked with analogous acquired or mnemonic changes in the cerebellar cortex.

These two differences—absence of variety of stimuli and absence of memory—would partly account for the uniform character of the cerebellar cortex and the probable, though disputed, absence of long association connections between different cortical areas. Impulses entering are spread out over a certain area, "corticated," and sent out. The mechanism of association would appear to be provided more in the successive spreads through afferent nuclei and their common diffusion, such as the olivary, pontile and partly segmental spinal nuclei, on the cortex and the successive integrations by the nuclei in the efferent paths.

As far as the cortex is concerned, it appears to do the same thing to all its afferent impulses whether they come from the pallium or more directly from the receptors.

In the interpretation of the cerebellum and of the cerebellar cortex, one is met at the outset by a barrier, i. e., the lack of knowledge of the respective origins of the climbing and mossy fibers. Most of the discussions of this question have been based on the assumption that these fibers have different origins. I should like to question this assumption and advance the counter assumption that all masses of gray matter sending fibers to the cerebellar cortex send both kinds of fibers. A third alternative is the one recently advocated by Lorente de Nó, i. e., that the climbing fibers arise from the cerebellar cortex itself, probably from the cells of Purkinje, such cells forming a separate category. Some of the cells of Purkinje would thus be cortical association neurons. Such a view opens up a new and interesting vista. It is

difficult to see, however, how such cells can be different from the others as far as reception goes. There seems to be every evidence that all Purkinje cells are in connection with granule cell and basket cell axons. It is possible, though, that only certain cells receive climbing fibers. This only emphasizes the need, pointed out elsewhere, for a thorough areal study of the cerebellar cortex, all methods being used. These important questions can be determined by an intensive study of cerebellar cortical areas which receive fibers from one or another source although this would perhaps, at the most, only differentiate paleocortical from neocortical areas. As far as I know, however, no such areal differences have been definitely made out. In fact, one striking thing about the cerebellar cortex is its apparent uniformity in all parts and in all animals in which it is present, though it exhibits a progressive development in higher mammals. Nevertheless, a careful study of the cerebellar cortex should be made. Another method would be a study of axonal secondary degeneration, silver stains being used after various afferent cerebellar connections are cut.

When the process of "cortication" is viewed, one finds the following: a possible direct return or a shorter cortical circuit provided by the climbing fibers, and a longer one provided by mossy fibers, granule cells and basket cells and involving a spread both lengthwise and transversely along the laminae and also a short spread by collaterals of Purkinje cell axons. There also appears to be a reciprocal or back-lash circuit involving the granule and van Gehuchten cells. Analogies to this arrangement may be seen in the dorsal gray horn, the gelatinosa corresponding to the granule layer, as pointed out, I think, by Johnston years ago; also, perhaps more remote analogies are present in the pallial cortex and elsewhere.

Attention might be called especially to the spread by Purkinje collaterals. Axon collaterals originating near the cell body are of nearly universal occurrence in the central nervous system, suggesting, perhaps, some universal function. It is obvious that a blocking of the impulses passing along the axon by a synaptic change, for instance, in a refractory period might cause a collateral overflow into adjacent neurons of similar connections, but whether this would mean a compensatory "evening" of the impulses, or some reciprocal innervation, it is difficult to tell. In this and in the overlap of the previously mentioned afferent diffusions may lie, in part, the mechanism for the restoration of function after various lesions. "Centers" in cortexes may be merely predominating areas for certain receptions or emissions, with the possibility of spread by the overlaps to other such centers, provided the lesion is not too extensive.

Whether the cerebellar cortex is merely doing, on a vaster scale to all impulses of a certain character and in a more precise way, what is

done elsewhere in various parts of the gray matter of the central nervous system, or whether there is something peculiar to the requirements of cerebellar regulation is difficult to tell, but it is obvious that here as elsewhere one is witnessing and will witness a rapprochement between the neurologist, with his crude explanations made to fit clinical observations, and the general neurophysiologist, with his all-or-none principle, his refractory periods, summations, facilitations and inhibitions, but with incomplete knowledge of clinical problems and sometimes of neuro-anatomy.

Returning to the question, Why a cerebellar cortex at all? one or two theories of cerebellar activity may be considered.

While it is said that there is a trend against the tonic theory of cerebellar function, there is much in the theory that appeals to me, following the lines of thought expressed by Walshe some time ago—in seeking the explanation of various phenomena seen in cerebellar lesions as fundamentally due to diminished tone—although possibly modified along lines of thought expressed by Mills, Weisenburg, Hunt, Tilney and Pike.

In general, in discussing the function of any coordinating mechanism, there are two questions to be discussed: a topographic or somatotopic one—the coordination of spatially separate parts, and a qualitative one—the change in the state of each part. In cerebellar disorders, one sees a difficulty in maintaining postures requiring complicated and delicate readjustments, in which I would include standing and maintaining equilibrium during walking and various acts, in maintaining posture necessary for the performance of certain movements and acts—fixations, and in the even performance of movement itself—steadiness and eumetria (the motofacient component of Tilney).

In all these, there are two elements: One is not exactly, perhaps, the maintenance of an even tone but also the continuous adjustment of tone in maintaining positions of unstable equilibrium and during the performance of movement in the moving part itself. The other, the topographic element, is seen in the proper localization of these tonic adjustments in the parts requiring them in the various activities indicated above. It is the latter, in the various muscles involved, that is expressed in the concept of synergy. The synergy can, however, consist only of the proper amount of simultaneous tonic or kinetic activity or both in the performance of some movement or movements. The change or adjustment of tone during an isolated movement, in the moving part, would perhaps involve only the antagonist muscle or muscles, though not necessarily so. A working conception on the basis of increase of both might be easier from an anatomic point of view as not requiring so complicated a mechanism, as well as explaining cerebellar asthenia. The

presence of this tonic component during movement is seen in the difference between the movements of an atonic tabetic person and a hypertonic parkinsonian person. The difference between motion in atonic and tonic conditions is much like that between the motions of flail-jointed dolls and those with friction joints. The parts of the latter stay in positions in which they are placed, and passive movement is different. A closer analogy would be when the tonus of the various muscles is represented by rubber bands which do not during passive (i. e., imposed from without) movement either stretch or relax. The question is complicated by the combinations with active shortening of some of the muscles to extracerebellar influences, but the point is that the cerebellar flow into the muscles is equal for all. The same might apply to the more static mechanisms, as standing and fixation. This would enable one to construct a tentative anatomicophysiologic theory which postulates an even diffusion by the cerebellar cortex of tonic impulses spread over appropriate cortical areas by the general but not necessarily universal diffusions of afferent fibers from the various subcerebellar nuclei—whether conveying direct proprioceptive stimuli or stimuli from more indirect sources. The topographic element is determined by the distribution of the afferent fibers on the cortex, the equalization of the tonic modification by intracortical processes which seem well adapted in a general way to secure this result. Or the view may be taken that the cerebellar influence is to equalize the tonus of the lower reflex mechanisms. This would require a varying intensity of the cerebellar influence. Lorente de Nó has well said that the cerebellum does not so much perform reflexes as it exalts the activity of some and depresses the activity of others. This view might require a variable outflow which would not necessarily result in an equalization of tonus. In any case, cerebellar activity might be roughly compared to the governor of an engine with its automatic, continuously acting, plus-and-minus influence, thereby maintaining, so to speak, a constant tonic pressure. Allusion has already been made to the discrepancy between rubral lesions which appear to cause hypertonicity and cerebellar lesions which appear more usually to tend to result in hypotonicity. This has been accounted for by others by assuming that the cerebellum has an inhibiting influence on the ruber. According to this view, the cerebellar influence might be either to increase or to decrease tone in various circumstances and also to do both simultaneously in an areal way. In this, as in the purely inhibiting view, one encounters the inhibition mystery, but if the pallial cortex can simultaneously "voluntarily" shorten some muscles and relax others (e. g., "letting" an elevated arm fall) why cannot the cerebellar cortex "involuntarily" and areally raise tonus in some parts and decrease it in others? It is obvious that if such a plastic adjusting activity is

attributed to the cerebellar cortex the variability, ambiguity and contradictions seen in the results of various experiments and also in clinical cases may be thereby explained, and it is also obvious that for such a complex function as is involved in any one of these views, a cortex is necessary. If, however, one had to choose between a uniform increase and a uniform diminution of tone as an expression of cerebellar activity, it would be the former which would be chosen, for certainly cerebellar disturbances admit best of explanation by a general hypotonia, as shown in the article by Walshe already alluded to. It may be objected that the "tonic" change of a moving part is practically a clonic change. I am not sure, if this is true, that it constitutes any more than a nominal objection, unless it would seem to postulate a motor character to the cerebellum, which experiment would seem to show that it does not possess. When it is considered how this more abrupt change is, so to speak, secondarily linked with movement, this objection might not hold. It has never been entirely clear to me just why only those points of the pallial cortex in most direct connection with lower motor neurons yield motor reactions. Stimulation of other points, of course, would pass through more synapses to reach such direct connections, but it does not seem entirely clear why this should make the difference, although this may be the explanation. It may be that better methods of stimulation may be found which will advance this method of research.

If the view of Lorente de Nó regarding association Purkinje cells is true and they provide a linkage of distant cortical areas, topographic coordination might be regarded as furnished by them instead, or partly instead, of the subcortical diffusion, or such connections might simply distribute the already integrated cortical adjustments of one area to another.

In any case, as previously remarked, the pallial cortex with its qualitatively different areas on both the receptive and the emissive side and its complicated association mechanism appears to be different fundamentally in its arrangements from the cerebellar cortex. In the latter, afferent diffusions, probably all essentially alike qualitatively and differing only topographically, are present. After "cortication" by the area to which they diffuse, they pass out in successive concentrations and split into paths to the various infracerebellar motor systems, and perhaps the supracerebellar pallium, whose activities they regulate.

REPERCUSSION

One point that should be considered in relation to cerebellar activity is the question of what might be called peripheral repercussion. The activities of various reciprocal innervations obtaining between various parts of the central nervous system might be termed central reper-

cussion. By peripheral repercussion is meant the explanation of a central function by peripheral stimuli set in activity by the central nervous system and returning to the activating center.

Undoubtedly, in all activities but especially in proprioceptive ones there is a continuous, so to speak, alternating back and forth flow of impulses from and to and from periphery to center. As each new posture is assumed, a fresh set of proprioceptive stimuli stream in and lead to further adjustments. What is meant here, however, are more extreme theories. The James-Lange theory of emotion, which identifies emotion with visceral and perhaps other "sensations" aroused by mnemonic cortical conditioned reflex activity, is one. Another is the Watson theory of identification, as I understand it, of various "mental" operations with the cortical reception of proprioceptive impulses from various muscles, especially of speech, set in activity by cortical mechanisms. Undoubtedly, the visceral or proprioceptive reverberation may play an important part in emotion or in mental activity, respectively, and in the latter case certainly in the acquisition of skilled movements. However, the extreme forms of such theories appear to attempt unduly to minimize or even to do away with the importance of the pallial associative mnemonic mechanisms. Similar questions arise in connection with the explanation of the reinforcement of the deep reflexes and of various abnormal associated and involuntary movements by muscular effort of some other part, also by emotion. Into this question there enters the question, mentioned earlier, as to the independent activity of lower reflex circuits. It is easy to see, for example, how an increased action exerted by a higher center on such a lower circuit, if summated with other afferent impulses received by such a lower center would cause a change in the muscles which would in turn start a new reflex activity through the lower circuit. A repercussion theory in the case of the cerebellum would be that the cerebellar adjustments take place after the reception by it of a peripheral proprioceptive stimulus, initiated by impulses from some other part of the central nervous system. Of course any new adjustment—change of posture and movement—arises from a bed, or basis, or foundation of an existing proprioceptive equilibrium maintained by proprioceptive stimuli, but the palliocerebellar connections render it possible to imagine that—in the case of, say, a voluntary movement—it need not wait until the movement is initiated and it receives the proprioceptive stimuli resulting therefrom, but that its activity is initiated by the pallium concurrently with the pyramidal movement. Further work on reaction times might be useful here. It is also evident, from what has been said, that unless there is found a more ample spread on the cerebellar hemispheres of direct proprioceptive stimuli from the body, especially the extremities, one must imagine that the proprioceptive control of part at least of the neocere-

bellum reaches it only via the pallium. It might be worth while here to deepen the clinical observations still further by paying still more attention to whether the cerebellar disturbance, for example, affects more standing and walking or more individual voluntary movements—in passing, how many look for the ataxic tongue?—and also whether there is any distinction between the voluntary skilled movements and the involuntary emotional ones. Is it or is it not true that cerebellar ataxia is increased by emotional states and by simultaneous voluntary effort exerted on other parts, as is the case with the so-called abnormal involuntary movements, and how is it affected by attention?

Edinger long ago pointed out that cerebellar development in various vertebrates varies with their complication of delicate adjustments in moving through space. An application of this to man would emphasize especially the requirements of his upright posture. The fact, however, that cerebellar disturbances manifest themselves equally in various voluntary activities not involving general bodily posture would seem to show that the emphasis is more on the finer regulation and coordination of all movements and postural adjustments. It also shows that it is the development of the various mnemonic or neokinetic activities rather than only those involved in the upright posture itself, the latter correlated with the activities described above, to be sure, which has necessitated, so to speak, the great development of the cerebellum in man.

COMMENT

In brief, it is found that the source and nature of many of the impulses projected on the cerebellum by its various afferent paths and the way in which such impulses are affected in their passage through subcerebellar synapses are not known; in certain important details, the areal projection of these various afferent paths on the cerebellar cortex is not accurately known; important critical details of cerebellar cortical structure still exist, the ignorance of which also prevents one from forming a conception of its activity—an added obstacle being the inadequate knowledge of general neurophysiology and its formulation in structural terms; many important details as to the distribution of the cerebellar outflow are not known. Consequently, one does not know how far there is a topographic representation and how far there is a blending or nonblending of different forms and combinations of activity such as postural and kinetic, older and newer, voluntary and involuntary and emotional. Nor does one know how far the organ functions more as a directly reacting or indirectly reacting mechanism.

It is also evident that probably less is known about neocerebellar than about paleocerebellar connections.

VASOMOTOR AND PILOMOTOR MANIFESTATIONS

THEIR LOCALIZING VALUE IN TUMORS AND LESIONS OF THE SPINAL CORD: A REPORT OF THIRTEEN VERIFIED CASES*

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Clinical means that permit more accurate localization of lesions of the spinal cord are of extreme value. This paper calls attention to a new sign and its significance as shown in thirteen verified cases, recorded herewith. It has proved of great value in determining the exact level for operation when sensory manifestations were incomplete or misleading, and in each case the upper level of vasomotor manifestation corresponded to the upper level of the lesion disclosed by operation or the roentgen ray.

VASOMOTOR MANIFESTATIONS

The patient should be examined after a period of rest in bed and should be free from clothing which may produce constriction or irritation to the skin. The patient is placed in diffuse daylight of moderate intensity, the possibility of shadows being avoided, the covering is quickly removed, and the entire body scanned carefully for vasomotor manifestations. This may best be determined at a distance of from 3 to 6 feet from the patient, and careful attention should be paid to the presence of areas of hyperemia, which are suggested by a broad band of flushing about the chest or abdomen, demarcated above by the normal flush of the skin.

The direction of this band of vasomotor manifestation is in striking accord with the segmental zones of Henry Head. Several variations of the phenomena may exist, and all have been found to be of equal localizing significance, indicating an abnormal instability in this area. The skin areas above and below the line of demarcation may vary in texture and in the degree of light reflection, if the patient is observed from an angle such as from the head or from the foot of the bed. Diffuse daylight should be used in all instances, as artificial light frequently gives rise to misleading effects.

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The actual line of demarcation may assume a feather-edged appearance and may be no larger than the diameter of a piece of twine extending in a serpentine fashion at right angles to the axis of the body until the axillary line is reached, and then coursing upward at an angle of about 30 degrees toward the spinous processes. The zone of demarcation in cases in which the history indicates a long standing involvement of the cord may simulate the difference between a slight sunburn and normal skin, so that the area below the level of the lesion appears to be slightly pigmented, and this effect may extend over a large area of skin below the line of demarcation, giving the appearance that the patient had been overexposed to the weather.

The examiner will be struck by the rapid evanescence of the vasomotor reaction in the zone under observation. During the initial period of observation a flushing should appear at the level noted, similar to that seen in slight hyperemia following the application of heat to the skin. If the patient is exposed to a draught or cold, it may be noted that this area blanches rapidly, giving rise to an apparent ischemia or to a zone distinctly white in comparison with the normal areas above.

Three patients in this series were negroes, with deep pigmentation of the skin. Even in them the vasomotor level was distinctly demarcated, being especially noticeable by the "light reflex" when viewed from an angle, the area above giving the soft, oily appearance of normal skin; whereas, in the zone of vasomotor disturbance, the surface was more velvety, apparently having lost some properties of secretion.

In eleven cases of this series, I was able to trace out, before making the sensory examination, the vasomotor line with a pencil on the surface of the skin. The level of vasomotor demarcation was found to coincide exactly with the first signs of sensory change, which began with this line and became increasingly more definite below this level.

When the apparent vasomotor demarcation has been determined by the eye, confirmation of this zone can be intensified by heat. A large bath towel wrung out in hot water should be spread over the entire chest and abdomen, including the areas above and below the suspected line of demarcation. After permitting this application of moist heat to remain for about five minutes, the skin surface is again exposed, and it will be noted that a zone of hyperemia has been induced by the heat both in the normal areas and in the areas below the line of demarcation.

Careful observation may now reveal such intensification of the features of the skin areas below the level of demarcation, as seen in figure 8, that photography of the point of change in skin reaction is possible. The area of skin below the line of demarcation shows fluctuating vasomotor properties, hyperemia rapidly fading into ischemic zones and then reappearing in characteristic blushing. After the nor-

mal skin areas have returned to their original color, a distinct zone of hyperemia at the upper line of demarcation may persist for several hours.

After a suitable time has elapsed following the vasomotor determination, the careful testing by a blunt object to determine the upper zone of hyperalgesia gives a line that exactly corresponds with the upper level of vasomotor demarcation and may appear one or more segments above the true zone of objective sensory disturbance.

As is evident in case 4, the vasomotor manifestations are probably radicular, and are therefore of extreme value in giving the exact level of the upper root irritated by the lesion. As pain in the root is not always a symptom in tumor of the cord, the vasomotor level indicates the site of involvement of the root. Frequently, the segmental level of involvement of the cord demonstrates itself objectively, much lower on the skin areas than the symptoms in the sensory roots, as these roots in the lumbar cord may have to pass the lesion to gain their point of exit below. This sign, therefore, becomes of increasing value to the surgeon for properly localizing the level of operative intervention.

PILOMOTOR MANIFESTATIONS

The pilomotor reflex is obtained by flicking the skin with a cold towel, or by the use of an electric fan or a current of cold air when the skin has been moistened by a cold towel. Pilomotor manifestations may also be produced by a method recently suggested by Thomas.[1] Deep pinching of the skin and the trapezius muscle at the base of the neck will produce a homolateral reflex spreading rapidly down the same side to include the lower extremities. This is a test for function of the cord and may give the level of the lesion by failing to appear below the involved segment (case 12).

The cold towel method that I used often denotes the level of irritation of the root by the intensity of the reaction. It is a segmental reflex and has been noted to occur below the level of a transverse lesion.

In three cases of this series, the pilomotor manifestations appeared above the level of the lesion and ended abruptly at the line of demarcation seen on the skin, being entirely lost below this area.

REPORT OF CASES

CASE 1.—*Tumor of the spinal cord at the fifth thoracic segment on the left. Endothelioma. Operation with removal.*

History.—Dr. R. S. M., was admitted to the University Hospital, on the service of Dr. William G. Spiller, Aug. 31, 1926, complaining of pain in the back and abdomen, with numbness and weakness of the left leg and loss of pain sensation in the right leg. The patient had been well until 1921, when he began to have

1. Thomas, A.: Rev. neurol. **1**:767 (June) 1926.

pains around the heart and paresthesias in the precordial area on the left. In 1923, there were sharp pains in the abdomen, especially in the left epigastrium, which were thought to be due to adhesions from a former appendectomy. The pain was continuous and was increased by bending over. For a month before admission, he noted numbness on both sides of the abdomen extending into the lower extremities, more marked on the right side. Three months before admission he noted a loss of temperature sense in the right leg associated with burning pain in the outer aspect of the right foot. Constipation had been present for three months, but no loss of bladder function. Weakness of the left leg became sufficient to require the use of a cane in walking.

Neurologic Examination.—There was no loss of the sense of touch; the sense of pain was definitely impaired in the right leg below the knee with slight impairment bilaterally as high as the umbilicus. The sense of position was normal. The vibratory sense was entirely lost in the left lower extremity. The temperature sense was practically lost in the right leg, and as high as the ninth interspace on the trunk. There was great impairment on the left side of the trunk as high as the eighth interspace. There was less involvement of the left lower extremity. There was definite weakness and spasticity in the left leg (fig. 2).

The reflexes were normal in the upper extremities; the abdominal reflexes were lost; the cremasteric reflex was normal on the right and diminished on the left. The patellar reflexes were greatly increased on each side; the achilles reflex was increased on the left and normal on the right. There was a positive Babinski sign on the left and a negative one on the right. There was no ankle clonus.

Laboratory Tests.—Roentgenographic studies and studies of the blood and urine gave negative results. The Queckenstedt test showed a partial block.

Vasomotor Tests.—The vasomotor level showed a clear-cut line of demarcation at the fifth thoracic segment bilaterally, with increased pigmentation of the skin areas below the level of the lesion. The vasomotor zone of demarcation corresponded exactly with the upper level of hyperalgesia and was in the area of the precordial pain. A definite point of tenderness was found over the third dorsal spinous process (figs. 1 and 2).

Diagnosis.—The condition was diagnosed as tumor of the spinal cord at the fifth thoracic segment on the left.

Exploratory Laminectomy.—Operation was performed by Dr. Grant, Oct. 6, 1926, and disclosed an intradural and extradural endothelioma below the level of the fourth thoracic lamina with pressure on the cord at the level of the fifth and sixth thoracic segments, involving the root of the fifth thoracic segment on the left (fig. 3).

CASE 2.—*Tumor of the spinal cord at the level of the fifth thoracic segment on the left. Endothelioma. Operation with removal.*

History.—Mrs. W. M., aged 53, was admitted to St. Agnes Hospital, Philadelphia, on the service of Dr. D. J. McCarthy, Oct. 2, 1926, complaining of numbness and weakness of the lower extremities. She had been well until sixteen months before, when, while standing beside the mantelpiece in her room, she suddenly fell to the floor because of weakness of both lower extremities. There was incontinence of the bowels and the bladder. She had been confined to bed and had been unable to walk without aid or assistance since. The numbness and weakness persisted, but the bladder symptoms improved, until five weeks before admission, when she again noted an increase in the symptoms, with loss

of bladder and rectal control. During the five weeks previous to admission there was rapid progression of symptoms, with gradual increase in the numbness, so that at the time of admission it extended as high as the waist. The weakness in the lower extremities became marked and she was unable even to move the limbs while in bed. Three months before admission she noted a sharp and shooting pain over the left side of the chest in the region of the precordium.

Neurologic Examination.—There was no definite weakness of the muscles of the lower portion of the trunk. The lower extremities showed almost complete paralysis. Spontaneous reactions of defense were present.

The reflexes in the upper extremities were active and equal. The abdominal reflexes were lost bilaterally. The patellar reflex was greatly exaggerated on the right and increased on the left. The achilles reflex was slightly increased on both sides. There was bilateral abortive clonus, with bilateral Babinski and Oppenheim signs.

The sense of position was lost in both low extremities. The sense of vibration was lost below the thorax. The temperature sense was lost below the ninth thoracic segment. Tactile sense was lost below the fifth thoracic segment on each side. Pain sense was lost below the fifth segment on the left and the seventh on the right (fig. 5).

Vasomotor and Pilomotor Manifestations.—A zone of vasomotor reaction was present over the chest and posteriorly at the lower angle of the scapulas. There was definite hyperemia in the lower two thirds of the breast on the left side. Posteriorly, the vasomotor line of demarcation seemed to be slightly lower on the right than on the left. The zone of hyperalgesia corresponded exactly with the vasomotor manifestations. There was distinct tenderness to pressure over the third dorsal spinous process (figs. 4 and 5).

The Queckenstedt test showed a complete block.

Exploratory Laminectomy.—Operation was performed by Dr. Fay, Nov. 9, 1926, and disclosed an endothelioma on the left side of the cord, at the level of the fifth thoracic segment, which was removed (fig. 6).

Comment.—The vasomotor level in this case was of distinct localizing value and accurately outlined the upper level of the lesion, as shown by operation.

CASE 3.—*Tumor of the spinal cord. Second to sixth segments. Subdural lipoma over the dorsal surface of the cord. Operation with partial removal and recovery.*

History.—Miss M. G., aged 21, was admitted to the Episcopal Hospital, Philadelphia, on the service of Dr. George Wilson, Oct. 25, 1926, complaining of pain over the heart, with numbness and weakness of the lower extremities. She had been well until six weeks before, when she noted pain around the region of the heart and over the left side of the chest. She was confined to bed for one week but noted on sitting up that pain was present, especially when she bent forward. Five weeks before admission she returned to work and noted weakness in the lower extremities when attempting to board a trolley car. The weakness progressed so that within four days she was unable to walk.

Neurologic Examination.—Vibration sense was lost in the right lower extremity and as high as the costal margin. It was impaired on the left to an equal level. Position sense was lost in both lower extremities. Pain sense was impaired in both lower extremities and as high as the eleventh thoracic segment, more marked on the left; slight impairment of pain sense was noted as high as the sixth thoracic segment bilaterally. Temperature sense was greatly impaired

in the right lower extremity and as high as the twelfth thoracic segment on the right. It was bilaterally impaired as high as the sixth thoracic segment. There was a definite point of tenderness over the fourth dorsal spinous process. A definite level of hyperalgesia was obtained at the fifth thoracic segment, corresponding to the zone of root pain (fig. 9). Pain in the precordial area was accentuated by coughing and sneezing.

A Queckenstedt test showed definite evidence of spinal block.

There was marked paralysis of both lower extremities with reactions of defense.

Bilateral clonus and Babinski sign were present. The abdominal reflexes were greatly diminished, and there was an increase in all the deep tendon reflexes of each lower extremity.

Vasomotor and Pilomotor Manifestations.—A definite zone of vasomotor reaction was noted on the left side, extending from the third thoracic segment to about the level of the tenth rib. Intensification of this zone by heat produced a unilateral flushing, as seen in figure 8, confined to the left side and extending from the upper level of the scapulas to fade away in the lower portion of the chest. This wide zone of vasomotor reaction was difficult to interpret, but operation revealed ample cause for its wide distribution, though the unilaterality of the sign is still a matter of conjecture (fig. 7).

Exploratory Laminectomy.—The operation, performed by Dr. Fay, extended from the second to the sixth dorsal segment of the cord. A subdural tumor was disclosed, extending over the dorsal aspect of the cord throughout the area of exploration (fig. 10). An attempt to remove the tumor failed to disclose any line of demarcation between the tumor and the substance of the cord. A portion of the tumor was taken for microscopic study and proved to be lipoma, similar in character to that reported recently by Stookey.[2]

CASE 4.—*Tumor of the spinal cord of the third and fourth thoracic segments. Intramedullary, probably glioma. Operation with decompression.*

History.—W. L., a man, aged 54, colored, was admitted to the University Hospital on the service of Dr. William G. Spiller, Aug. 30, 1926, complaining of pain in the region of the right shoulder, numbness in the right lower extremity, and weakness of the right leg. He had been well and occupied with farming until May, 1926, when he first noted pain below the right shoulder blade, which was aching in character and gradually extended around to the anterior surface of the chest on the right side. Shortly after the onset of pain, the right leg began to feel numb and stiff. Two months before admission, weakness occurred in the right leg, so that on admission the man required a cane for walking. Constipation began one month before admission and was associated with difficulty in urination. At the time of admission, the patient complained of a girdle sensation and peculiar drawing feelings in the left leg.

Neurologic Examination.—Pain sense was lost on the right as high as Poupart's ligament; above this was a zone of hyperalgesia; in the lower abdomen, from the costal margin to a point just above the nipple on the chest, pain sense was greatly diminished; pain sense was lost on the left side, including the lower extremities and abdomen, and over the thorax to a level just below the nipple. Vibration sense was impaired in the right lower extremity and as high as the thorax; it was normal on the left. Sense of position was normal in

2. Stookey, Byron: Intradural Spinal Lipoma, Arch. Neurol. & Psychiat. **18**:16 (July) 1927.

both lower extremities. Touch sense was impaired up to the level of the nipple line, bilaterally. Temperature sense corresponded to the levels of pain sense but was apparently more greatly disturbed in the left lower extremity than in the right (fig. 12). There was an area of tenderness to deep pressure over the fourth and fifth spinous processes.

There was definite weakness of the right lower extremity.

The abdominal reflexes were lost; the patellar reflexes were greatly exaggerated, especially on the right; the right achilles reflex was exaggerated, the left normal; there was no Babinski sign and no clonus.

Laboratory Examination.—The laboratory and roentgen-ray reports were essentially negative. A Queckenstedt test was positive for a partial block.

Vasomotor and Pilomotor Manifestations.—A definite zone of vasomotor demarcation was noted in this case even in the presence of the deep pigmentation of the skin characteristic of the patient's race. The skin appeared oily and normal above the zone of demarcation, but there was a distinct tendency toward a velvety and dull luster to the skin below. The zone of demarcation on the right appeared at a level just above the nipple; on the left it was just below the nipple. The addition of heat to accentuate the vasomotor zone made the line of demarcation even more evident. The application of cold to the skin produced a pilomotor reflex that was intense in the area above the zone of vasomotor demarcation; it ceased abruptly at this line and did not extend below into the level of sensory disturbance (fig. 11).

Diagnosis.—The condition was diagnosed as tumor of the spinal cord on the right at the level of the fourth and fifth thoracic segments.

Exploratory Laminectomy.—The laminae were removed by Dr. Frazier, Dec. 18, 1926, over the second, third, fourth and fifth thoracic segments. Marked enlargement of the cord was disclosed in segments three and four; the process was cystic and probably gliomatous. A decompression was made and recovery followed (fig. 13).

Comment.—At the operation it was necessary to section the third and fourth thoracic roots in order to explore the anterior surface of the cord. Following this operative procedure, the patient presented a new level of vasomotor manifestations; the zone of vasomotor demarcation had risen to include the third thoracic segment, extending out on the arm sufficiently to include the axillary region and over the upper border of the chest. The old line of vasomotor demarcation was still evident by slight increase of pigmentation of the skin, but this was not so marked as formerly.

It would appear therefore, that the vasomotor reaction noted on the skin in this case was probably a radicular pressure phenomenon with loss of vasomotor control to the dermatome involved.

CASE 5.—*Intramedullary tumor of the cord at the level of the fifth, sixth and seventh thoracic segments. Exploratory laminectomy with decompression and incision of the tumor mass. Recovery.*

History.—Miss E. K., aged 17, was admitted to the Orthopedic Hospital, Philadelphia, on the service of Dr. T. H. Weisenburg, Sept. 26, 1925, complaining of weakness and numbness of the lower extremities for the past six months. She had been apparently well until five years before. At that time the mother noted staggering in gait. The patient frequently fell or stumbled when going upstairs. Examination by a physician disclosed a curvature of the spine, and a cast was applied for three months. Symptoms entirely disappeared for four years. One year before admission she again noted staggering, with coldness and numbness in the feet,

which gradually extended upward until at the time of admission the area was as high as the waist. The weakness of the lower extremities had also increased. There was a girdle sensation in the lower portion of the chest. Bladder symptoms began four years before, with frequency and urgency, and persisted throughout the illness. There was no incontinence.

Neurologic Examination.—There was bilateral loss of the abdominal reflexes. Marked spasticity of the lower extremities was present, more pronounced on the left. The patellar and achilles reflexes were exaggerated bilaterally. Ankle clonus was present on both sides, more marked on the right. Bilateral Babinski and Oppenheim signs were elicited. The sense of vibration was lost in both lower extremities. Sense of position was lost in both lower extremities. Temperature sense was lost on the right as high as the fourth intercostal space and greatly diminished on the left as high as the fifth intercostal space. Tactile sensation showed marked diminution in the lower extremities as high as the costal margin. Temperature sense was involved in a similar degree and area as the pain sense (fig. 15). A level of hyperalgesia corresponded to the upper level of vasomotor manifestation. A Queckenstedt test was positive.

Vasomotor Manifestations.—A vasomotor level was determined on the left side over the chest in about the third and fourth interspace (fig. 14).

Diagnosis.—The condition was diagnosed as tumor of the cord at the level of the fifth and sixth thoracic segments, at the level of the fourth root.

Exploratory Laminectomy.—Operation was performed by Dr. A. P. C. Ashhurst, Dec. 2, 1925, and disclosed an intramedullary tumor of the cord extending from the fifth to the seventh thoracic segments. Decompression was made, with closure, and recovery followed. A second operation was performed by Dr. A. P. C. Ashhurst and Dr. Fay, Feb. 4, 1926, with exposure of the tumor and the normal area of the cord above. Cordotomy was performed, with section of the anterolateral columns above the tumor mass for relief of pain. Incision into the tumor revealed a gelatinous center which was thought to be glioma. Further decompression was secured with recovery (fig. 16).

Comment.—The tumor was found to be situated in the region from the fifth to the seventh thoracic segments at the first operation; it had extended slightly higher at the second operation and involved the fourth thoracic segment.

The vasomotor sign still persists (fig. 14).

CASE 6.—*Gunshot wound of the spinal cord with destruction of the sacral portion. The bullet entered the midline at the level of the first lumbar spine. The roentgen ray disclosed it lodged at the level of the ninth dorsal vertebra.*

History.—Mr. W. D., aged 24, was admitted to the Philadelphia General Hospital on the service of Dr. George Wilson, Dec. 1, 1926, complaining of paralysis of both lower extremities following a gunshot wound of the spine. He had been well until Oct. 16, 1926, when he was shot by a policeman and was removed to the hospital in an unconscious condition. The bullet had entered the middle of the back at the level of the first lumbar spine, taking an upward course. The roentgen ray revealed the bullet lodged at the level of the ninth dorsal vertebra in the region of the liver (fig. 19). The patient had complete flaccid paralysis of the lower extremities, with marked wasting and atrophy of all muscle groups of the legs and around the buttocks. There was complete anesthesia below the second lumbar segment, including the sacral area, with incontinence of bowels and bladder.

Neurologic Examination.—Symptoms of a transverse lesion of the cord at the second lumbar segment, with destruction of the sacral and lumbar portion

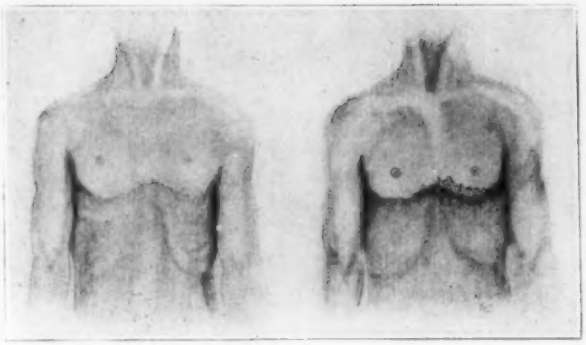

Figure 1

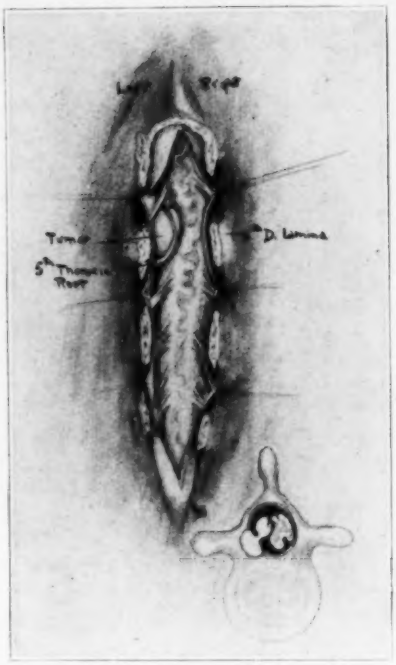

Figure 3

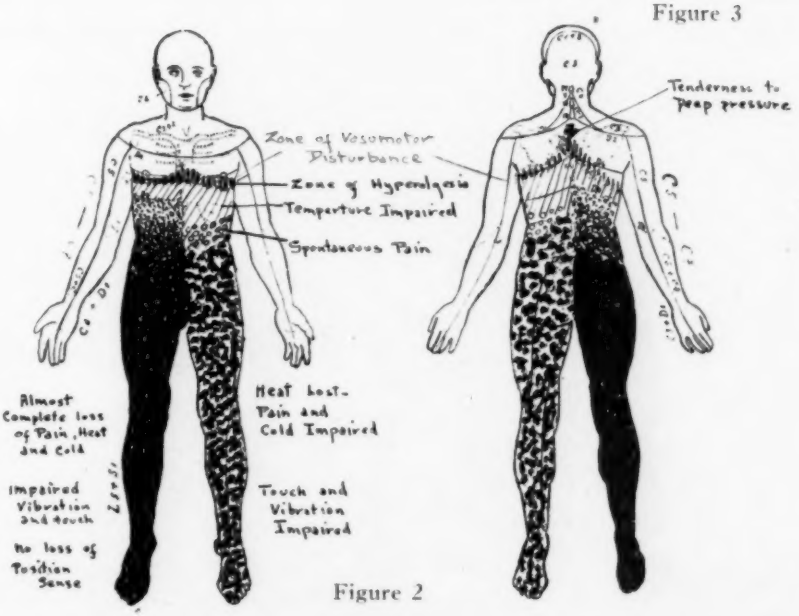
Zone of Vasomotor Disturbance
Zone of Hyperalgesia
Temperture Impaired
Spontaneous Pain
Tenderness to Deep pressure
Almost Complete loss of Pain, Heat and Cold
Impaired Vibration and touch
No loss of Position Sense
Heat lost. Pain and Cold Impaired
Touch and Vibration Impaired

Figure 2

Fig. 1 (case 1).—Change in pigmentation of the skin below the vasomotor level of demarcation. On the left, the patient as he appeared at the time of the initial examination. On the right, the vasomotor level intensified by the hot towel test. The tumor was an endothelioma removed from the fifth thoracic segment.

Fig. 2 (case 1).—The upper level of the zone of hyperalgesia, which corresponds almost exactly with the vasomotor level. The level of spontaneous pain and distinct sensory change led at first to the consideration of a lesion at a much lower level. The point of tenderness to deep pressure on the back was situated just above the tumor.

Fig. 3 (case 1).—Extradural and intradural endothelioma of the cord involving the fifth thoracic root and the sixth segment on the left. The tumor was entirely removed at operation. The vasomotor level proved to be of exact localizing value.

Figure 4

Figure 6

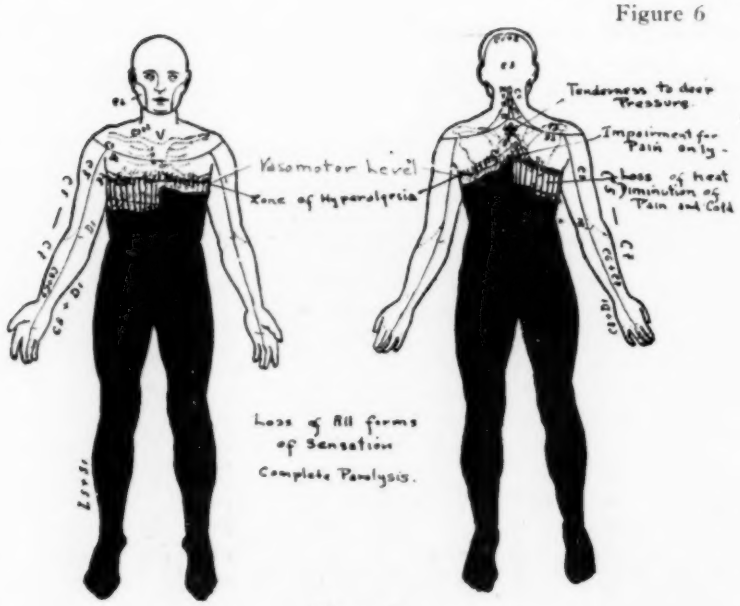

Figure 5

Fig. 4 (case 2).—The vasomotor level as seen after the application of heat to the skin. The tumor, as seen in figure 6, involved the fourth and fifth thoracic roots on the left.

Fig. 5 (case 2).—In this case, the sudden onset of symptoms and the absence of definite root pains with profound sensory disturbance led to the diagnosis of myelitis. The distinct level of vasomotor manifestations, the corresponding level of hyperalgesia and the positive Queckenstedt sign led to an exploration. The tumor was found and removed from below the point of deep tenderness to pressure shown on the back.

Fig. 6 (case 2).—Endothelioma of the cord involving the fourth and fifth thoracic roots on the right and the fifth thoracic segment of the cord. The tumor was completely removed at operation. The vasomotor level definitely established the upper level of the lesion.

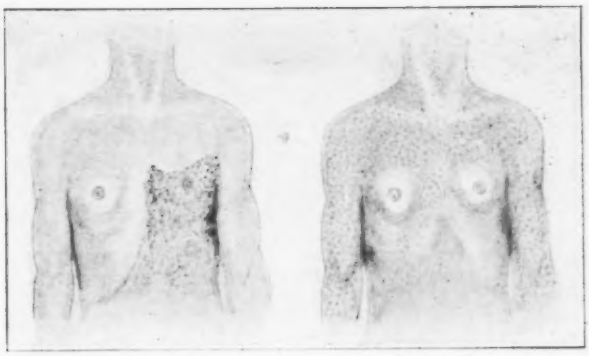

Fig. 7 (case 3).—The vasomotor response to heat shown on the left was almost entirely unilateral. The pilomotor response showed an even higher level and did not extend below the vasomotor line of demarcation on the left. The tumor, as seen in figure 10, was a large subdural lipoma of the cord covering the dorsal aspect with slightly more marked involvement of the left side. The wide zone of vasomotor response and the high level of the pilomotor disturbance corresponded closely to the large number of segments of the cord involved.

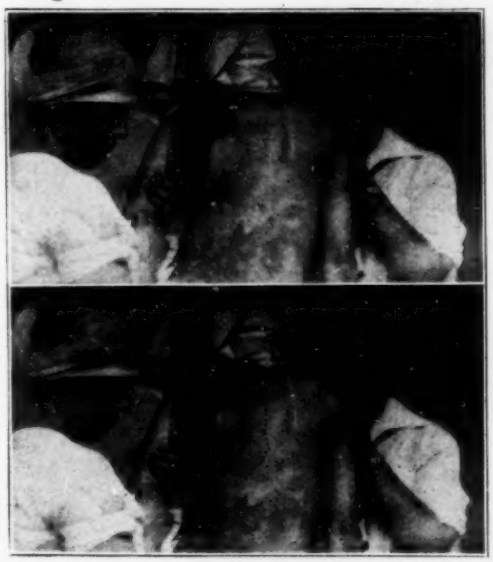

Fig. 8 (case 3).—The wide zone of vasomotor response after the application of a hot towel to the back. In the lower illustration, the zone has been marked out to make apparent what is difficult to portray photographically. The zone includes the third thoracic segment extending onto the arm and into the axilla, also down the back to the sixth segment. In this case, the response was entirely confined to the left side. Identical photographs.

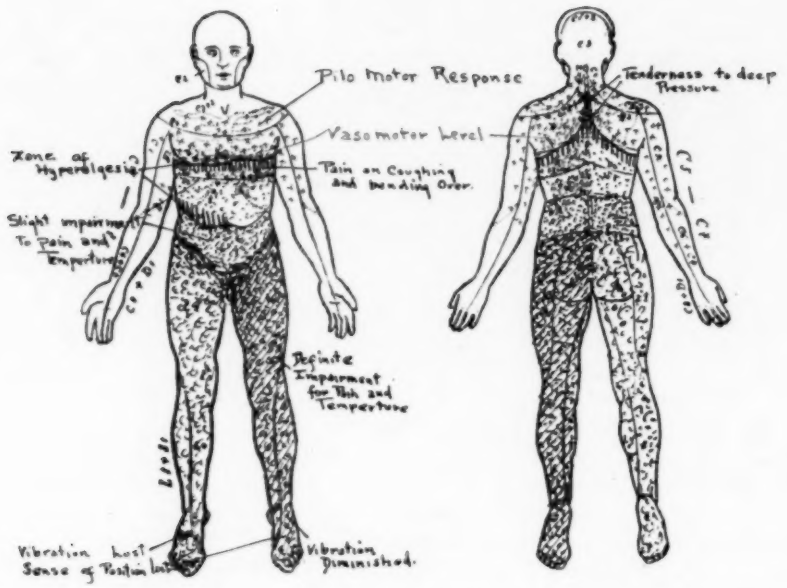

Fig. 9 (case 3).—The sudden onset of symptoms, the early sensory changes and the wide zone of vasomotor change led to some uncertainty as to the nature and extent of the lesion. The positive Queckenstedt test and the area of deep tenderness to pressure on the back were of assistance in diagnosing the lesion. The abrupt level in which the pilomotor responses ceased coincided with the upper level of the vasomotor change and the zone of hyperalgesia and thus determined the level of operative exposure.

Fig. 10 (case 3).—Appearance of subdural tumor, extending over the dorsal aspect of the cord throughout the area of exploration.

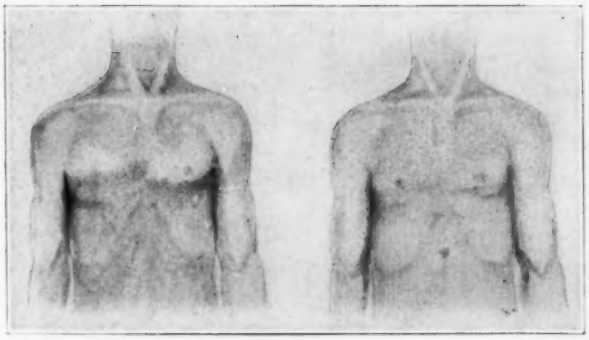

Figure 11

Figure 13

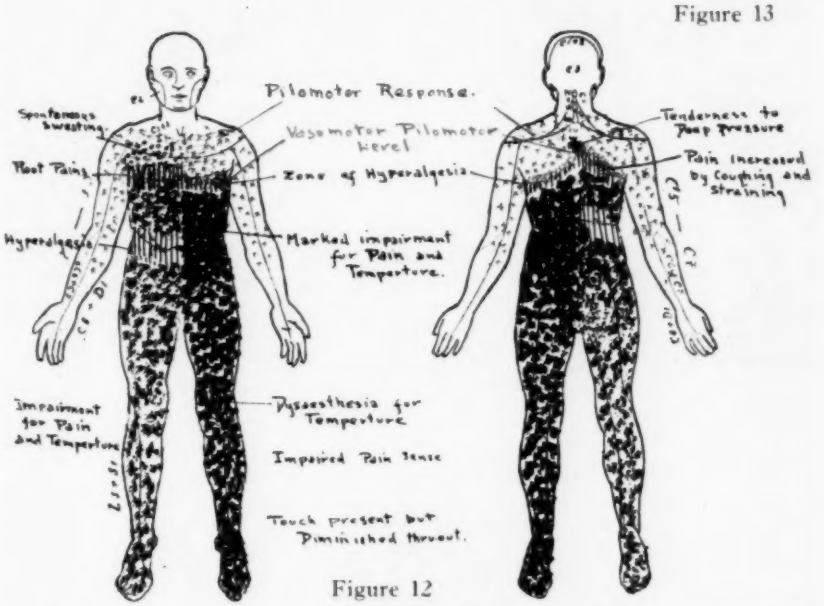

Figure 12

Fig. 11 (case 4).—The sharp line of vasomotor and pilomotor demarcation was striking in this case. The pilomotor response ceased abruptly at the level at which the vasomotor manifestations began. The tumor was found to be intramedullary and involved the third to the fifth thoracic segments. At operation the third and fourth thoracic roots were cut to give an opportunity to view the anterior surface of the cord. It was noted after operation that the vasomotor line had become symmetrical and had advanced to the level of the shoulder girdle, also including the axilla. It would appear from this observation that the vasomotor manifestations were due to loss of function of root fibers in the skin areas supplied.

Fig. 12 (case 4).—The vasomotor and pilomotor responses gave a definite and clearcut level which corresponded to the upper level of hyperalgesia and root pain. The pilomotor manifestations were active above the level of the sensory changes but absent below. The vasomotor changes became apparent at the level of the lesion and extended below but not above. The area of spontaneous sweating over the right upper portion of the thorax should be noted. The tumor was found to be an intramedullary growth extending into the third thoracic segment and slightly above.

Fig. 13 (case 4).—Marked swelling of the cord disclosed at operation, thought to be intramedullary glioma, partly cystic. The lesion was most marked in the fourth and fifth thoracic segments but extended to the third. The vasomotor level was distinct at the level of the fifth thoracic segment on the left and at the level of the fourth on the right. The increased sweating over the right upper part of the chest extended as high as the third segment, as represented by the skin area on the chest and in the axilla. The vasomotor level proved to be at the point of greatest involvement of the cord.

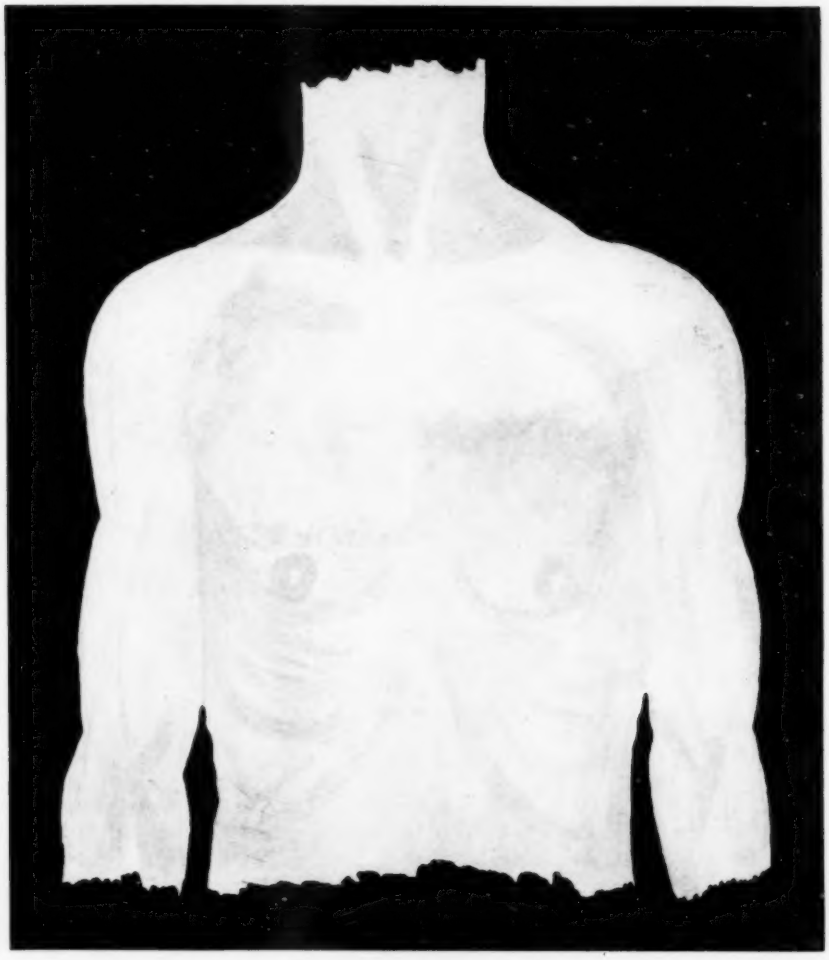

Fig. 14 (case 5).—The skin manifestations as seen on the patient without the aid of heat or cold. The change in the skin surface is slight and would almost escape detection if not viewed carefully and at a short distance from the patient. The application of cold following heat or blowing on the moist skin of the patient will often be enough to bring out a quick vasomotor response. The longer application of heat will maintain the skin hyperemia for a considerable time.

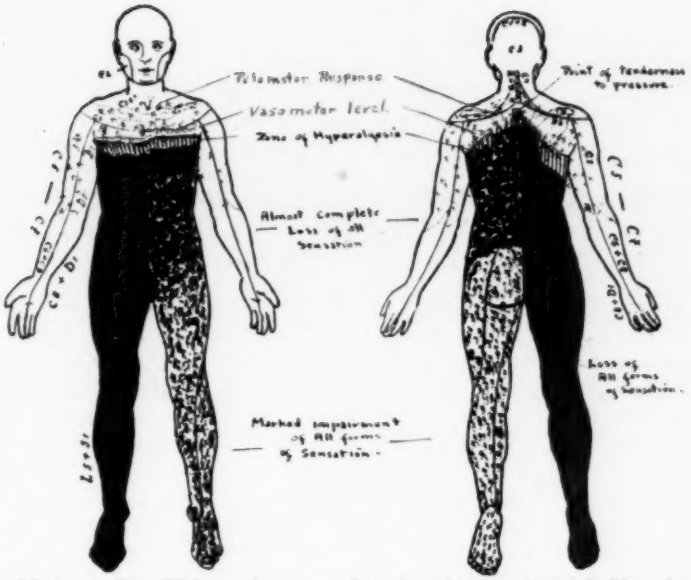
Vasomotor Response
Vasomotor level
Zone of Hyperalgesia
Almost complete Loss of all Sensation
Point of tenderness to pressure
Loss of all forms of sensation
Marked impairment of all forms of Sensation

Fig. 15 (case 5).—This patient was found to have a large infiltrating tumor of the cord, probably a glioma, extending from the fourth to the seventh dorsal segments. The vasomotor level was distinct at the upper zone of sensory change. The pilomotor response was diffuse but more marked above the level of the tumor than below. The symptoms of vasomotor and pilomotor manifestations have persisted throughout the year following exploration and decompression.

Fig. 16 (case 5).—Intramedullary glioma extending from the fourth to the seventh thoracic segments of the cord. The vasomotor and pilomotor manifestations accurately established the upper level of the lesion.

Fig. 17 (case 6).—The first figure to the left shows the dusky appearance of the skin below the upper level of the lesion as seen without means of intensification. The next figure represents the response of the skin to heat and the following one to the pilomotor response to cold. The intensification of this response in the zone of hyperalgesia over the abdomen and the absence of the manifestation at the level of beginning definite sensory change should be noted. The last figure shows the intense ischemia of the skin to ice with the appearance in this patient of a line like a white cord at the upper level of the vasomotor manifestations. It is interesting to note that a second level of vasomotor manifestation existed on the right, as seen in the first figure to the left, at the level of definite sensory change in the first lumbar segment. It was thought that the bullet had definitely damaged the cord at this level and had also injured the lower thoracic roots that pass the first lumbar segment to find their points of exit below the corresponding vertebrae.

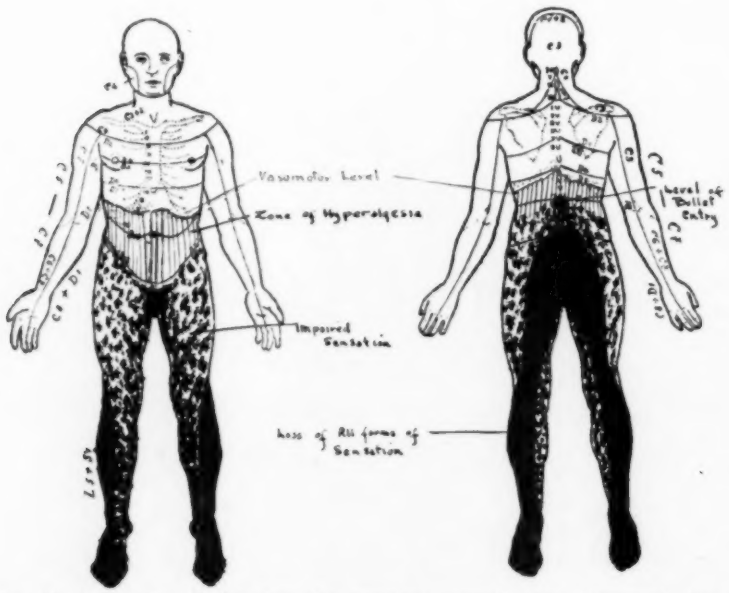

Fig. 18 (case 6).—Following the gunshot wound that destroyed the sacral cord, definite sensory changes were present to the level of the first lumbar segment. A definite vasomotor level was well established at the level of the eighth thoracic dermatomere. Careful testing showed a distinct zone of hyperalgesia with slight sensory changes. The bullet was located by the roentgen ray at the level of the ninth dorsal vertebrae.

below this level, were disclosed. There was complete anesthesia and paralysis with loss of patellar and achilles reflexes, and marked atrophy. From the level of the eighth thoracic dermatomere on the lower portion of the chest to Poupart's ligament there was hyperalgesia, associated with spontaneous root pains in this area. Below Poupart's ligament, beginning at about the first lumbar segment, there was marked impairment to all forms of sensation on each side (fig. 18). There was loss of bladder function with anesthesia of the scrotum and penis, and incontinence of feces.

Diagnosis.—The condition was diagnosed as destruction of the sacral and lower lumbar cord, by gunshot wound, causing traumatic myelitis involving the roots as high as the eighth dorsal.

Vasomotor and Pilomotor Manifestations.—There was a definite line over the anterior surface of the body and also toward the posterior lateral aspect of the abdomen, where a slight change in hyperemia of the skin was evident. The upper level of the line reached a point just under the costal margin and in about the eighth dorsal cutaneous segment. Vasomotor and pilomotor reflexes in this region could be produced by irritation to the skin, which gave an evanescent fluctuation of hyperemia and ischemia. At the upper line of vasomotor demarcation the objective sensory changes became evident. Above this line sensation was normal, but when a blunt object was drawn over the surface of the skin, hyperalgesia was immediately encountered at the level of the vasomotor disturbance. Hyperalgesia was present over the abdomen to the level of the first lumbar segment.

Application of heat to the abdomen and chest produced marked hyperemia in the zone of hyperalgesia; that is from the eighth thoracic to the first lumbar segment. In this zone, there was marked flushing following the application of the hot towel with a definite upper line of demarcation at the eighth dorsal segment. Cold applied to the skin produced a marked pilomotor reflex in the zone of hyperalgesia, as well as above, so that the only demarcation between the pilomotor reflex above and below the level of the lesion was manifest by a more intense reaction of the skin below the eighth dorsal segment and extending down to the first lumbar segment (hyperirritability). Above the eighth dorsal segment, in the normal response of the skin, the pilomotor influences were distinct but not as marked as in the zone of hyperalgesia (fig. 17).

There was an intense and marked reaction to cold which produced "goose flesh" that was distinct from that produced in the normal areas above. The application of cold in the form of ice produced a white line of ischemia along the upper level of demarcation. This line was so distinct and definite that it could be marked out on the abdomen and closely followed the upper line of vasomotor evanescence. The vasomotor demarcation exactly coincided with the upper level of sensory changes. Furthermore, the line as accentuated by cold was identical with the line of demarcation as noted on the patient's abdomen before the application of either heat or cold and detectable at a short distance from the patient by the eye alone.

Comment.—In this case the upper level of sensory disturbance was anticipated and a line was drawn on the chest before the sensory examination was made. It was found to coincide exactly with the upper level of hyperalgesia, and, on roentgen-ray studies, the bullet was shown to be opposite the ninth thoracic vertebra so that involvement of the eighth thoracic root and those below seemed evident.

CASE 7.—*Tumor of the spinal cord at the first lumbar segment on the right extending 7 cm. upward to involve the ninth thoracic root. Sarcoma. Operation with removal.*

History.—F. W. B., aged 65, was admitted to the service of Dr. William G. Spiller, Dec. 27, 1926, at the University Hospital, complaining of difficulty in walking and pain in the back. The patient showed a spastic paraplegia, with bilateral clonus and Babinski sign. The sensory examination showed impairment of pain and temperature only in the lower extremities, with loss of vibratory sense to the costal margin. There was a wide zone of hyperalgesia extending from the costal margin to the inguinal region on each side (fig. 21).

The roentgen-ray examination of the spine showed well marked hypertrophic spondylitis of the lumbar vertebrae. The Queckenstedt test showed a complete spinal block. The spinal fluid was xanthochromic. Other laboratory examinations gave negative results.

Vasomotor Manifestations.—There was a definite vasomotor level at the eighth thoracic dermatomere over the upper part of the abdomen on both sides. There was a lower level on the right side in this case that was clear cut in the first lumbar distribution on the right. This area was pigmented and showed marked hyperalgesia. It was the area in which the patient referred much of his pain (fig. 20).

Diagnosis.—Although the sensory manifestations were not convincing and as they failed to show a clear cut level and as hypertrophic spondylitis was thought to explain many of the symptoms, the reliability of the vasomotor sign in other cases led to the diagnosis of a tumor of the cord on the right side involving the first lumbar segment and several roots above. It was thought that the lesion might be large because of the wide zone of vasomotor manifestations similar to that seen in case 3.

Exploratory Laminectomy.—Operation was performed by Dr. Grant, Feb. 9, 1927, and disclosed a large vascular tumor on the right side of the cord extending over several segments and involving the ninth thoracic to the first lumbar roots (fig. 22). The tumor was removed.

Comment.—The definite vasomotor manifestations in this case led to the correct localization and the decision for exploration. The intense vasomotor reaction on the right side at the first lumbar segment proved to be the point of greatest involvement of the cord.

CASE 8.—*Tumor of the spinal cord involving the eighth, ninth and tenth thoracic roots on the left. Melanotic sarcoma. Operation with partial removal.*

History.—Mrs. M. H., aged 45, was admitted to the University Hospital on the service of Dr. William G. Spiller, March 28, 1927, complaining of paralysis of the lower extremities and loss of sensation. Six months prior to admission, the patient had noted weakness in both legs, which had gradually increased until two months later she could no longer walk. Numbness in the legs was noted at this time. On admission, she showed a complete transverse lesion with sensory and motor loss as high as the ninth thoracic segment (fig. 24).

The Queckenstedt test showed complete block. Roentgenograms and other laboratory examinations gave negative results.

Vasomotor Manifestations.—There was a definite vasomotor level at the eighth thoracic dermatomere, slightly higher on the left. Below this was a band of pigmentation extending to the tenth on the right and to the eleventh on the left (fig. 23).

Diagnosis.—The condition was diagnosed as tumor of the spinal cord at the ninth thoracic segment.

Exploratory Laminectomy.—Operation was performed by Dr. Grant, April 26, 1927, disclosing a melanotic sarcoma at the tenth thoracic segment on the left, involving the eighth and ninth roots. There were many small dark patches scattered over the arachnoid (fig. 25). The bulk of the tumor was removed.

CASE 9.—*Tuberculous spinal caries involving the sixth thoracic vertebra with transverse myelitis. Albee bone graft with partial recovery.*

History.—Mrs. S. H., aged 51, was admitted to the University Hospital on the service of Dr. William G. Spiller, Dec. 25, 1926, complaining of paralysis of the lower extremities and incontinence. Four months prior to admission, she had noted weakness in both legs. This rapidly increased and was associated with numbness, which extended as high as the costal margin.

At the time of admission to the hospital, there was great weakness of the lower extremities, with exaggeration of the deep reflexes, clonus and Babinski sign. Sensory tests showed an almost complete transverse myelitis to the level of the seventh thoracic dermatomere. A zone of hyperalgesia extended to the sixth on the left and the fifth on the right (fig. 27).

Vasomotor Manifestations.—On initial inspection at the time of admission, a definite vasomotor level at the seventh thoracic dermatomere was noted, with an area of pigmentation below (fig. 26).

Palpation of the back disclosed a definite prominence at the sixth thoracic vertebra, with angulation and a point of tenderness to deep pressure just above.

Roentgenograms showed a destruction and a crushing of the body of the sixth thoracic vertebra (fig. 28).

Diagnosis.—The condition was diagnosed as Potts' disease of the sixth thoracic vertebra.

Operation.—A correction of the angulation and an Albee bone graft was undertaken, Feb. 24, 1927, by Dr. Gill. The patient later regained partial function of the lower extremities.

Comment.—The patient was referred with the diagnosis of tumor of the spinal cord; the slight angulation of the spine had not been noted. The vasomotor sign indicated the level of the lesion on initial inspection, and examination at this level of the back revealed the evident spinal deformity. Roentgen-ray studies made the following day disclosed the suspected vertebral disease.

In this case, the patient was a negro, and the level was seen without the aid of heat by standing at the head of the bed; the "light reflex" gave a soft, deep, velvety appearance below the level of the lesion as compared with the shining oily normal areas above.

CASE 10.—*Tumor of the spinal cord, involving the first lumbar root on the right with pressure on the sacral cord. Extradural sarcoma. Operation with partial removal.*

History.—H. L., aged 46, was admitted to the Orthopedic Hospital, on the service of Dr. Astley P. C. Ashhurst, Jan. 25, 1927, complaining of severe pains in the legs and in the right groin. Incontinence had developed, and there was increasing weakness in the lower extremities. Morphine was required for alleviation of the pain.

Sensory examination did not show a clear cut level. There was loss of sensation to pain in the sacral distribution but no disturbance of tactile, position or vibration sense. Slight hyperalgesia was noted over the lumbar distribution (fig. 30).

The reflexes in the lower extremity were absent on each side, and atrophy of the muscles of the legs and buttocks was present.

Vasomotor Manifestations.—In this case the vasomotor sign was striking and was confined to the first lumbar dermatomere on the right side only. This area was distinctly flushed, and the similarity to the condition in case 7 was so marked that a lesion of the first lumbar root was suspected; this, together with the sacral disturbance placed the lesion at the level of the first lumbar vertebra, catching the root of exit on the right. It was suspected that the lesion might be extradural, and this diagnosis was made at the time (fig. 29).

The Queckenstedt test was positive for a complete block, and the spinal fluid showed slight xanthochromia.

Diagnosis.—The condition was diagnosed as extradural tumor involving the first lumbar root and sacral cord.

Exploratory Laminectomy.—Operation was performed by Dr. Ashhurst and Dr. Fay, April 28, 1927, and disclosed a vascular tumor in the region of the first lumbar vertebra invading the spinal canal and the muscle bed without. The lamina of the first lumbar vertebra was found to be soft and infiltrated. The tumor lay on the right side of the cord, and a large portion produced pressure on the dura from without (fig. 31).

Comment.—In this case the vasomotor sign was of great value in giving the location of the tumor as well as its probable extradural position. Operation disclosed that the first lumbar root was involved in the tumor mass as it emerged from the dura. It is apparent that the interpretation of this sign of involvement of the root gave the level for operation in the lumbar region, whereas if the condition had been considered as a manifestation of involvement of the segment, a higher exploration would have been necessary.

CASE 11.—*Fracture dislocation of the first lumbar vertebra with destruction of the sacral and lower lumbar cord. Twenty-eight years' duration.*

History.—Mrs. B. B., aged 45, was admitted to St. Agnes Hospital, on the service of Dr. Leonard Averett, March 17, 1927, complaining of trophic ulceration of the right heel, total prolapse of the uterus and transverse myelitis of the lower lumbar cord. At the age of 17, the patient fell 30 feet from a balcony, sustaining a fracture dislocation of the first lumbar vertebra and a fracture of both femurs. For the past twenty-eight years she had walked on crutches, though there was complete paralysis of both lower extremities below the knees.

I saw the patient in consultation as to the advisability of hysterectomy in the presence of the neurologic manifestations.

The sensory examination showed a complete loss of all forms of sensation below the second lumbar distribution. There was complete paralysis of the lower extremities below the knees, with atrophy and loss of reflexes. Hyperalgesia was present in the area between the eleventh thoracic and the second lumbar dermatomere (fig. 33).

Vasomotor and Pilomotor Manifestations.—There was a striking yellowish pigmentation of the skin seen in the area between the eleventh thoracic and the second lumbar dermatomeres, inclusive (fig. 32). This gave the appearance of an apron, and close inspection showed that the skin was more deeply pigmented. This region corresponded to the zone of hyperalgesia.

The pilomotor reflex was spontaneous in this zone on algophoric stimulation, and was increased when pinched over the base of the neck (Thomas method).

These manifestations were considered to be due to irritation of the roots from a long standing traumatic pachymeningitis.

Roentgen-Ray Examination.—An old healed fracture dislocation of the first lumbar vertebra was shown by the roentgen ray (fig. 34).

Comment.—The patient was of extreme interest in that she was seen twenty-eight years after the initial lesion. The vasomotor sign was characterized by pigmentation of the skin, which was easily apparent. This conforms to the other cases in which the condition had been present for a period of months. The flushing noted in an early lesion (fourteen hours, in case 13) is replaced by pigmentation in the area later on.

CASE 12.—*Angioma of the spinal cord at the seventh, eighth and ninth thoracic segments. Exploration and decompression.*

History.—R. E., a boy, aged 19, was admitted to the University Hospital on the service of Dr. William G. Spiller, April 20, 1927, complaining of a heavy feeling in the legs, weakness and inability to walk without crutches. One year before admission, he noted difficulty in walking and a tendency to drag the feet. Six months later, it was necessary for him to use crutches, and shortly after this he noted incontinence of urine.

Sensory examination showed hyperalgesia at the seventh thoracic skin segment, extending down to the ninth. Pain and temperature sense were greatly impaired on the right side below the ninth thoracic segment. There was impairment of vibration, position and touch sense in both lower extremities and as high as the umbilicus. The reflexes were exaggerated, with bilateral clonus and Babinski sign (fig. 36).

Vasomotor and Pilomotor Manifestations.—The vasomotor level could be seen at the eighth thoracic dermatomere extending around the body. Some slight flushing at times occurred as high as the seventh dermatomere. There was a band of pigmentation below this level in the ninth to the eleventh thoracic skin segments (fig. 35).

The pilomotor reflexes were of great interest. When the skin at the base of the neck was pinched in the region of the trapezius muscle, a homolateral pilomotor response was brought out at once on the right side and extended to the lower extremity. When this test was applied to the left side, the pilomotor manifestation did not reach below the eleventh thoracic dermatomere. This was evidence that stimulation from the upper cord was interrupted on the left side at about the eleventh thoracic segment.

Diagnosis.—The condition was diagnosed as probably an intramedullary lesion of the spinal cord at the ninth thoracic segment.

Exploratory Laminectomy.—Operation was performed by Dr. Frazier on June 3, 1927. An extensive angioma of the cord was found, involving the sixth, seventh, eighth, ninth and probably the lower segments of the cord. Decompression without removal was performed.

As this is the only case in this series in which I did not view the lesion, no illustration is possible.

Comment.—The pilomotor manifestations in this case gave a clue to the intramedullary possibilities of the lesion and, together with the disturbance of pain and temperature sense on the right, seemed to indicate greater involvement of the cord on the left. I was unable to determine if the operative manifestations would bear this out.

CASE 13.—*Fracture dislocation of the eleventh thoracic vertebra; transverse myelitis and acute meningomyelitis. Exploration and decompression.*

History.—N. K., a man, aged 21, was admitted to my service at the Polyclinic Hospital, Jan. 1, 1926, with a broken back and complete paralysis of the lower extremities. The patient was found unconscious and paralyzed after receiving an injury in an unknown manner about fourteen hours previously.

Examination showed a complete motor and sensory paralysis below the waist, with a zone of hyperalgesia between the eighth and tenth thoracic dermatomeres (fig. 38).

Vasomotor Manifestations.—A definite band of flushing at the level of the umbilicus extended around the body and was fluctuating and feather-edged. Hyperalgesia was most intense at this level (fig. 37).

Roentgen-ray examination showed a fracture dislocation of the eleventh thoracic vertebra, with fracture of the lamina. The Queckenstedt test was positive.

Exploratory Laminectomy.—Operation was performed at once by Dr. Fay, and the lamina of the eleventh thoracic vertebra was found fractured and depressed, pressing deeply on the cord. After removal of the fragments, the cord was exposed and a localized intense swelling was disclosed (fig. 39). Decompression was effected.

COMMENT

In the thirteen cases reported in this paper, the vasomotor level was found to correspond exactly with the upper level of sensory disturbance and to indicate the highest point and extent of the lesion in the spinal cord. It has therefore proved of great value in accurately determining the level for operative intervention. It was found to be more reliable than the sensory level as determined in cases 1, 3, 7 and 10, and in all the cases studied it proved to be the most accurate sign for determination of the level. Four other important factors have been present in each case: (1) a definite history of involvement of the cord suggesting a localized lesion; (2) either a complete or partial Queckenstedt test was obtained in each case (except in case 6, which was caused by a gunshot wound the extent of which was evident from the roentgen-ray examination and in cases 9 and 11, in which roentgenograms disclosed the lesion); (3) careful testing for a zone of hyperalgesia at the upper level has shown it corresponded to the level of vasomotor change; (4) tenderness on deep pressure over the spinous processes was found near or just above the level of demarcation, as noted by the vasomotor change and hyperalgesia.

The reliability of the sign has been so definite that it was the determining factor in deciding on operative intervention in cases 2, 3, 7 and 10 in which the history of onset was so abrupt as to lead to the belief that tumor of the cord was unlikely. Since February, 1926, when the significance of this sign became apparent, twenty-three verified cases have been observed, thirteen of which are here reported. In all, the vasomotor level indicated the upper level of sensory demarcation and the highest point of involvement of the cord in cases of discrete lesions, such

as tumor of the cord, or in traumatic or vertebral disease. It has become possible with the four contributing factors noted to determine accurately the sensory level of disturbance of the cord by inspection of the skin on the patient before sensory determinations have been made. As yet, the observations have been confined to manifestations shown by lesions of the thoracic and lumbar cord.

The patients have been observed carefully for skin manifestations such as sweating and oiliness of the skin, hyperemia, ischemia and "goose flesh," with suitable tests to determine the value of each. It has been found that the vasomotor sign is most reliable and has been constant in all cases. The pilomotor manifestations are next in value but have not been found confined distinctly to the level of vasomotor or sensory disturbances in all cases.

At times two levels have been observed, the upper level determining the point of highest irritation of the root, with slight vasomotor changes in the skin below the point of involvement. The lower level has been characterized by pigmentation, which is definite and corresponds with the level of beginning objective sensory disturbance in cases in which the lesion has persisted for more than three months. The vasomotor levels may be close or several segments apart.

In attempting to determine an explanation for the manifestations noted regarding the pilomotor and vasomotor reflexes, the most comprehensive study dealing with this work was found in the recent monograph by Thomas[1] whose work on the sympathetic system satisfactorily explains the mechanism of the vasomotor and pilomotor disturbances in these cases.

Horsley utilized the vasomotor properties of sweating in his patients as a means of localization of a lesion of the cord. Thomas and his followers pointed out the values of vasomotor and pilomotor responses in vertebral and extravertebral lesions involving the sympathetic fibers. Foerster used the vasomotor response to stimulation of a dorsal root as a means of mapping out the segments supplied to the skin surface and thus determined the area of the dermatomere under consideration. The presence of this vasomotor sign in tumors or lesions of the spinal cord has been of distinct value in this series, demonstrating the highest point of involvement of the root by the lesion and thus indicating the exact level for surgical intervention.

CONCLUSIONS

1. A simple method for the accurate determination of the upper level of lesions and tumors of the spinal cord is outlined.

2. In thirteen verified cases which are reported the vasomotor line of demarcation has proved to be the most reliable sign; in each case it has determined the exact level of localization.

3. In all the cases a history of a lesion of the spinal cord was evident. The Queckenstedt test was positive for partial or complete block. The zone of hyperalgesia corresponded exactly with the upper level of vasomotor demarcation. A point of tenderness to deep pressure was found in each case over the spinous processes at the level of the lesion.

4. From the surgical standpoint, the vasomotor level indicates the root level for operative exploration and, in those cases coming to operation, has proved to be correct, even though the sensory tests in some would indicate a lower position of the lesion in the cord.

5. The vasomotor line of demarcation will frequently indicate the level of the lesion before the development of sensory or motor symptoms of focal value.

6. This vasomotor sign, when associated with a positive Queckenstedt test and a zone of hyperalgesia corresponding to the level of vasomotor demarcation (trauma and evidence of vertebral disease having been ruled out by history or roentgen ray) has been found so far only in lesions producing pressure on the spinal cord.

7. The earliest appearance of this sign was noted fourteen hours after injury to the cord (case 13) and the longest duration was twenty-eight years following a spinal lesion (case 11).

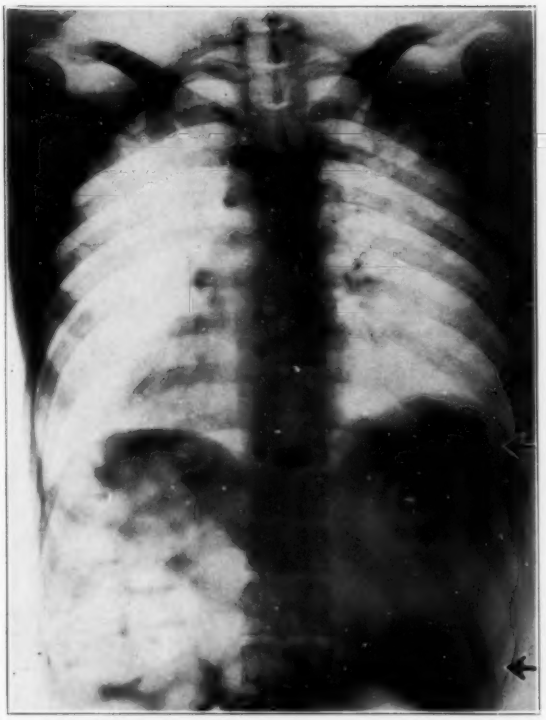

Fig. 19 (case 6).—Roentgenographic appearance of small fragments of lead at the tip of the first lumbar spinous process, indicating the point of entry of the bullet exactly in the mid-line (lower arrow). The bullet, indicated by the higher arrow, may be seen at the level of the ninth thoracic vertebra indicating its course and the area of cord involved, producing destruction of the cord below the first lumbar segment and injury to the ninth, tenth, eleventh and twelfth thoracic roots, passing toward their points of exit.

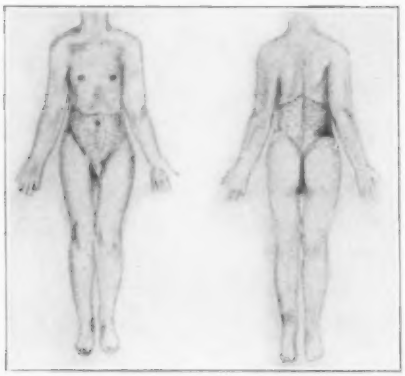

Fig. 20 (case 7).—Vasomotor manifestations seen clearly on inspection alone. The upper and lower levels in this case should be noted. The tumor was found to extend over three segments of the cord on the right side, being largest at about the first lumbar segment, but involving the roots of the ninth, tenth, eleventh and twelfth thoracic segments as well.

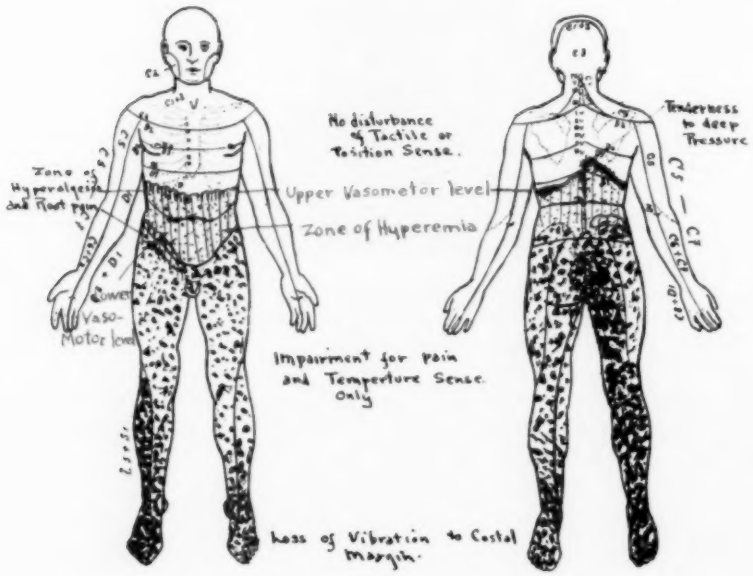
No disturbance of Tactile or Position Sense.
Tenderness to deep Pressure
Upper Vasomotor level
Zone of Hyperemia
Zone of Hyperalgesia and Root pain
Lower Vaso-Motor level
Impairment for pain and Temperture Sense Only
Loss of Vibration to Costal Margin.

Fig. 21 (case 7).—The sensory manifestations in this case were not clear cut and the finding of extensive hypertrophic spondylitis by the roentgen ray made the diagnosis of tumor of the cord difficult. The vasomotor sign and the positive Queckenstedt test led to the accurate localization and diagnosis of tumor of the cord, which was found and removed.

Fig. 22 (case 7).—A large elongated tumor on the right side of the cord removed at operation. The wide band of vasomotor manifestations on the right with clear cut upper and lower levels corresponded to the roots involved and proved the accurate localizing value of this sign. This should be compared with case 3.

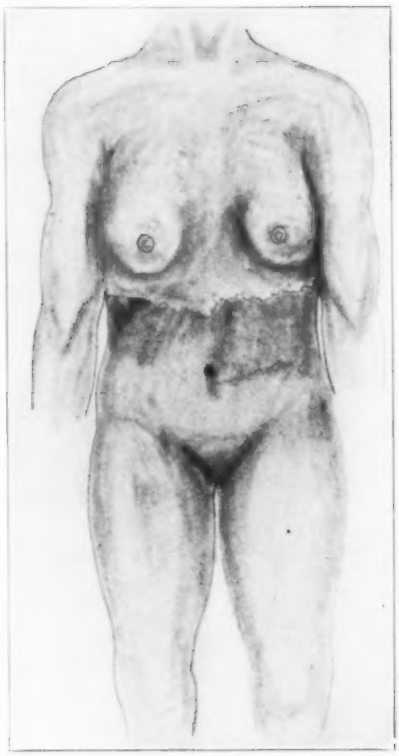

Fig. 23 (case 8).—The vasomotor manifestations seen in a colored patient, showing the intense pigmentation in the area of the vasomotor disturbance. An irregularly shaped tumor, extending over two segments of the cord on the left, was removed. It was found to involve the eighth, ninth and tenth thoracic roots.

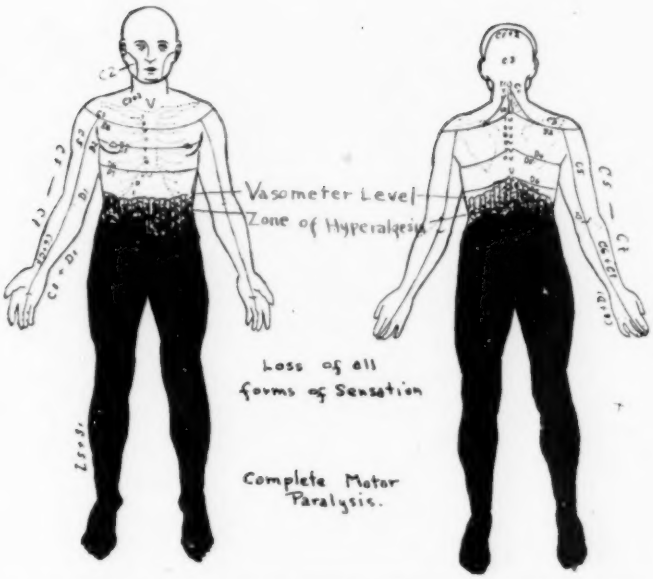

Fig. 24 (case 8).—The sensory manifestations, showing the corresponding zones of hyperalgesia and vasomotor manifestations.

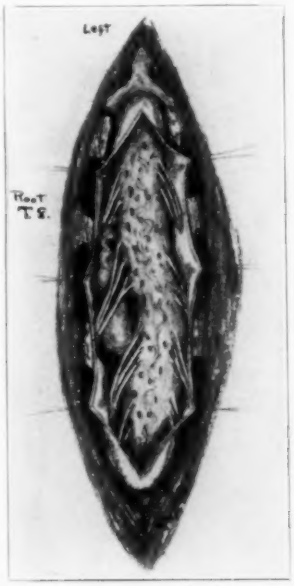

Fig. 25 (case 8).—A melanotic sarcoma, extending over two segments and involving three roots of the cord, which was removed at operation. Many small metastatic patches were noted on the arachnoid. The vasomotor sign accurately localized the level of the tumor at its highest point.

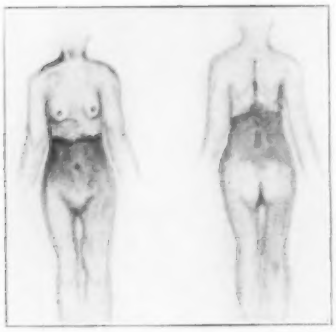

Fig. 26 (case 9).—The localization in this case was made by inspection alone immediately on admission of the patient. The vasomotor level was distinct at the seventh thoracic dermatomere with deep pigmentation below this level. There was a slightly higher vasomotor reaction on the right as high as the fifth thoracic skin segment. Careful examination revealed angulation of the sixth spinous process that had not been detected prior to admission. Roentgen-ray examination showed destruction of the sixth thoracic vertebra due to Pott's disease.

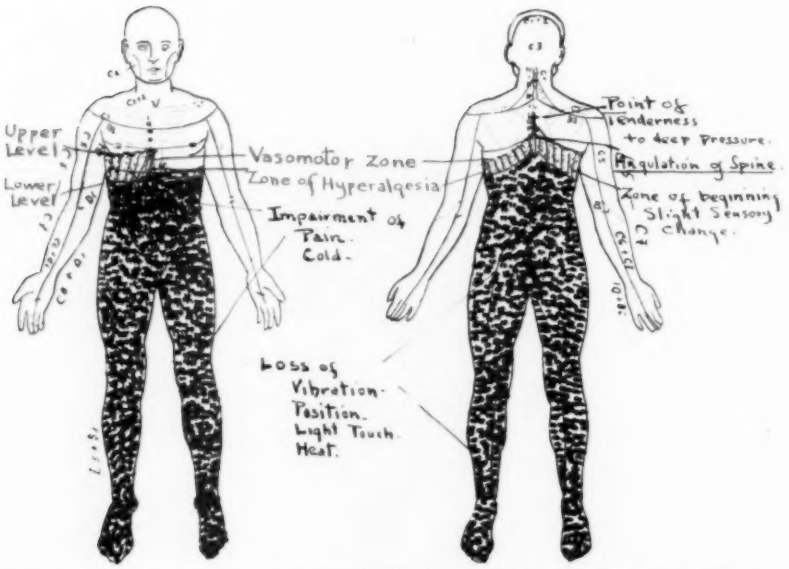

Fig. 27 (case 9).—The sensory manifestations at the time of admission, with a vasomotor zone indicating involvement of the fifth and sixth thoracic roots on the right, and the sixth on the left, and signs of a transverse myelitis below this level as indicated by sensory examination.

Fig. 28 (case 9).—Roentgenographic appearance of destruction and crush of the sixth thoracic vertebra due to Pott's disease. The fifth and sixth thoracic roots were probably involved in the destruction process or by pressure, giving rise to the vasomotor level noted in figure 26. The arrow indicates kyphosis.

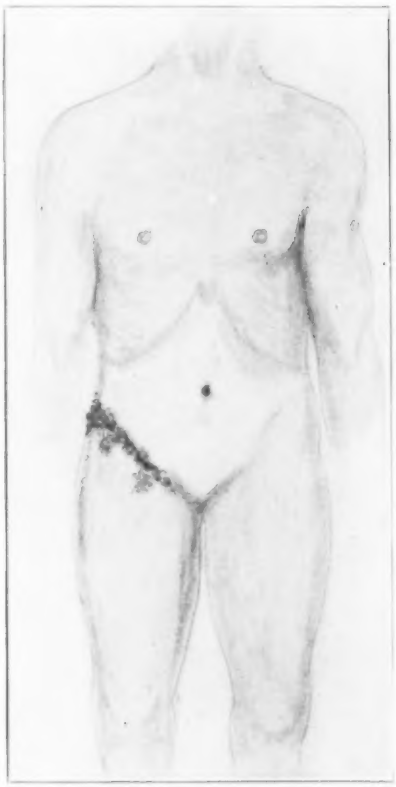

Fig. 29 (case 10).—The vasomotor manifestation in this case was confined to the first lumbar dermatomere on the right. As there was no other vasomotor disturbance, this led to the localization of the lesion at the first lumbar vertebra, catching this root at its point of exit in contradistinction to a lesion at the first lumbar segment of the cord which would have required exploration in the region of the eleventh thoracic vertebra, a point 3 inches higher. Operation revealed an extradural sarcoma involving the first lumbar vertebra with pressure on the conus. The vasomotor sign in this case was of distinct surgical value, indicating the proper level for exploration.

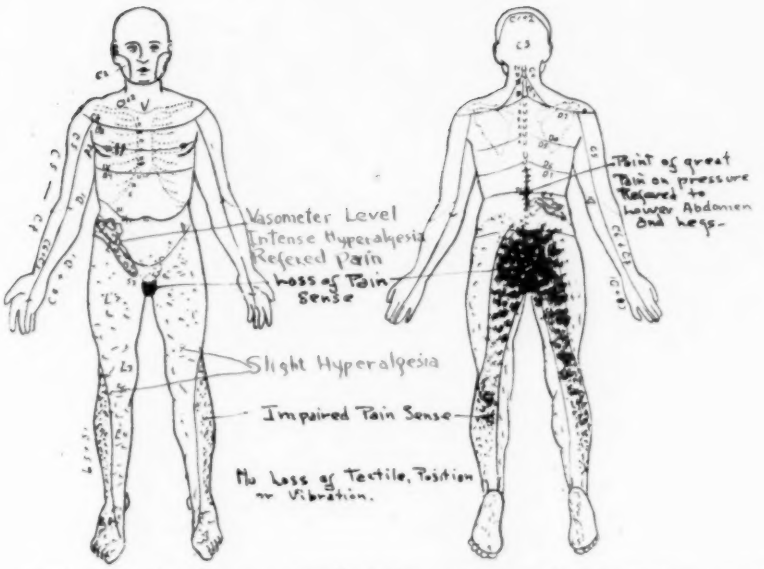
Vasometer Level
Intense Hyperalgesia
Refered Pain
Loss of Pain Sense
Slight Hyperalgesia
Impaired Pain Sense
No Loss of Tactile, Position or Vibration.
Point of great Pain on pressure Refered to lower Abdomen and legs.

Fig. 30 (case 10).—Sensory manifestations showing the beginning sacral involvement and the wide area of hyperalgesia. The sharp definition of the vasomotor level on the right should be noted. The lesion was considered one which produced pressure on the sacral cord and involved the lumbar roots as they passed this level to find their points of exit below.

Fig. 31 (case 10).—The tumor, as revealed at operation, proved to be an extradural sarcoma on the right side of the cord, involving the first lumbar vertebra and root with pressure on the cord within.

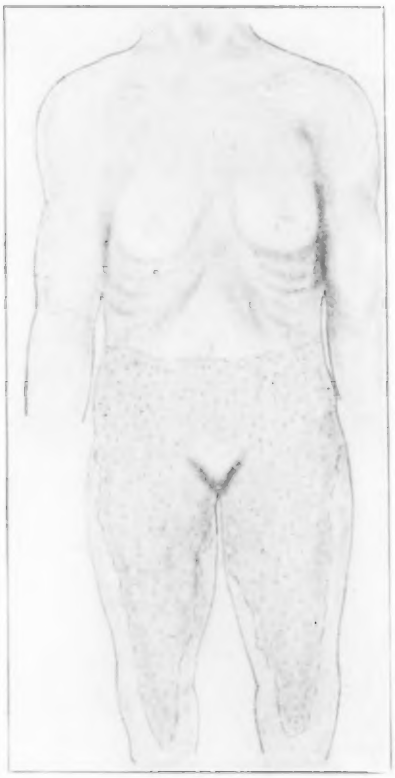

Fig. 32 (case 11).—The area of definite pigmentation and vasomotor manifestation was unique in this case as it followed a lesion of twenty-eight years' duration. It extended from the eleventh thoracic to the second lumbar dermatomere inclusive, showing marked hyperalgesia and hyperirritability to pain and temperature stimulation. Algophoric stimulation produced a spontaneous pilomotor response confined to this area.

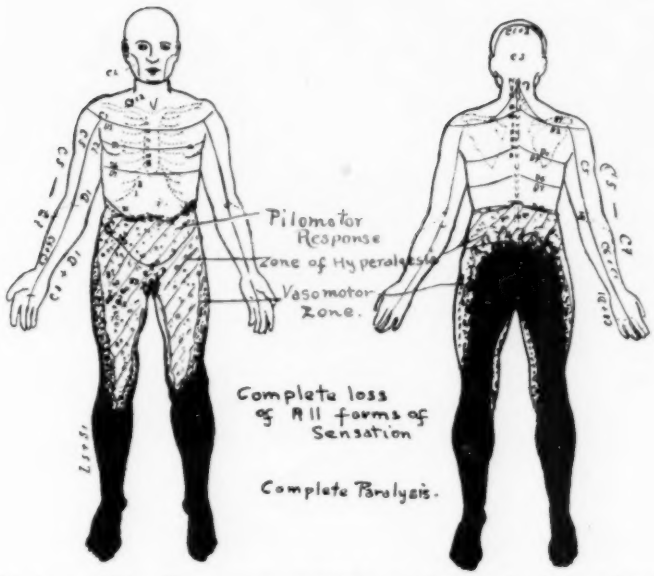

Fig. 33 (case 11).—Transverse myelitis at the level of the third lumbar segment due to an old injury and crush fracture of the first lumbar vertebra, producing irritation of the eleventh thoracic to the second lumbar roots, inclusive, with spontaneous pilomotor response confined to this area.

Fig. 34 (case 11).—The arrow indicates the crush fracture of the first lumbar vertebra of twenty-eight years' duration. The involvement of the roots as high as the eleventh thoracic may be due to a traumatic myelitis that involved the structures slightly above the site of fracture.

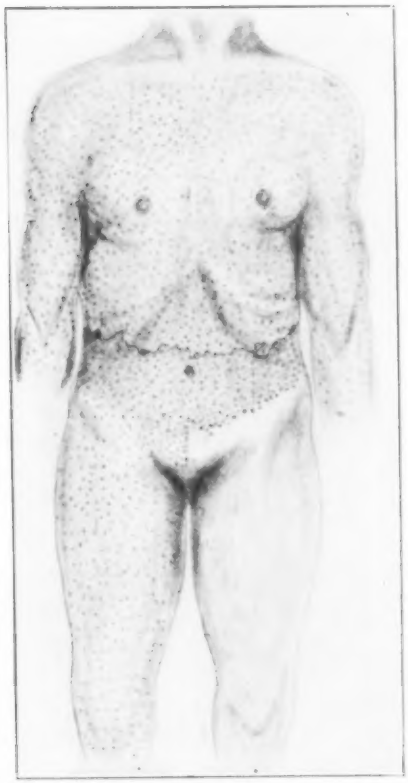

Fig. 35 (case 12).—Vasomotor and pilomotor manifestations in this case were of extreme interest. The vasomotor level was noted at the seventh thoracic dermatomere bilaterally with some pigmentation of the eighth, ninth and tenth segments on the skin. The homolateral pilomotor reflex produced by the Thomas method of pinching the skin and trapezius muscle at the base of the neck showed that the pilomotor response on the left did not extend below the eleventh thoracic segment of the cord, and therefore did not reach the skin areas below. This was interpreted as being due to a lesion that had interrupted the spinal pathways from above on the left side, and an intramedullary lesion was predicted. Operation disclosed a diffuse angioma of the cord extending over segments six, seven, eight and nine of the thoracic cord and probably lower.

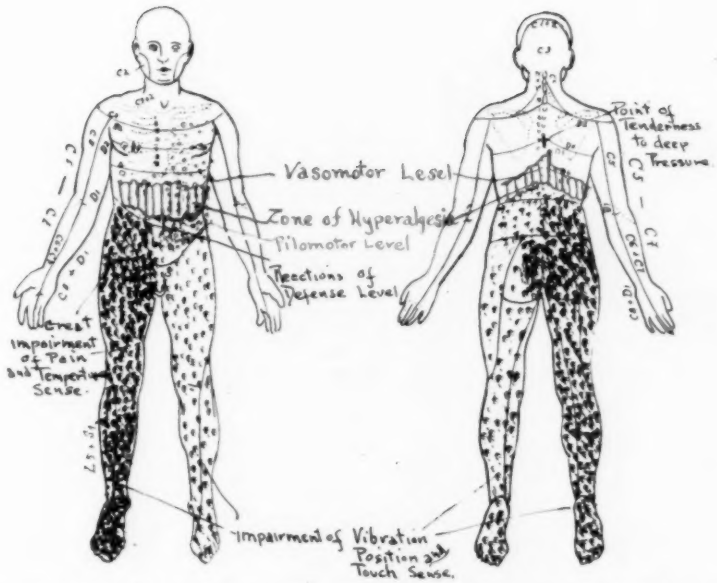
Vasomotor Level
Zone of Hyperalgesia
Pilomotor Level
Reactions of Defense Level
Point of Tenderness to deep Pressure
Great Impairment of Pain and Temperature Sense.
Impairment of Vibration Position and Touch Sense.

Fig. 36 (case 12).—The sensory manifestations in this case suggested more extensive involvement of the left side of the cord, as seen by the pilomotor responses and the pain and temperature impairment on the right. The pilomotor response (Thomas method) ceased at the eleventh thoracic dermatomere on the left. An angioma of the cord was found at operation.

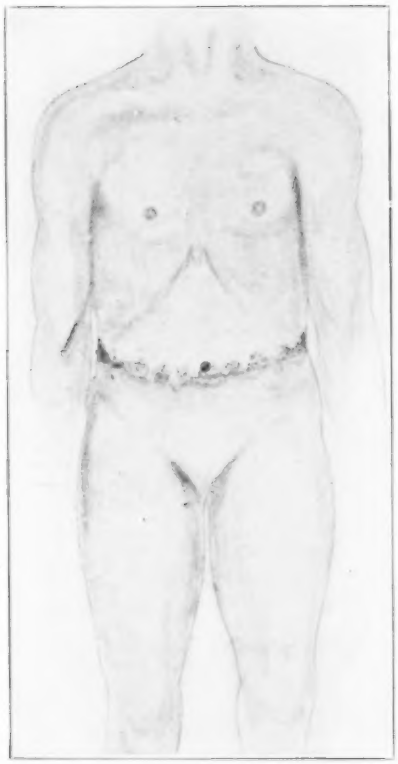

Fig. 37 (case 13).—Distinct vasomotor level noted fourteen hours after fracture dislocation of the eleventh thoracic vertebra with signs of transverse myelitis. This was the first case in which the vasomotor sign was noted (Jan. 1, 1926), and immediate operation disclosed a traumatic lesion of the cord at a level corresponding to the vasomotor sign.

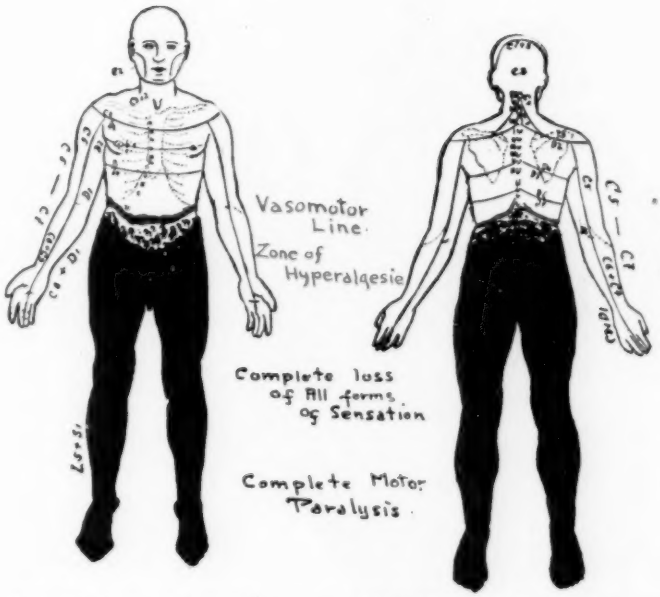

Fig. 38 (case 13).—Sensory manifestations of transverse myelitis with the vasomotor and hyperalgesic zone slightly higher than the level of complete anesthesia.

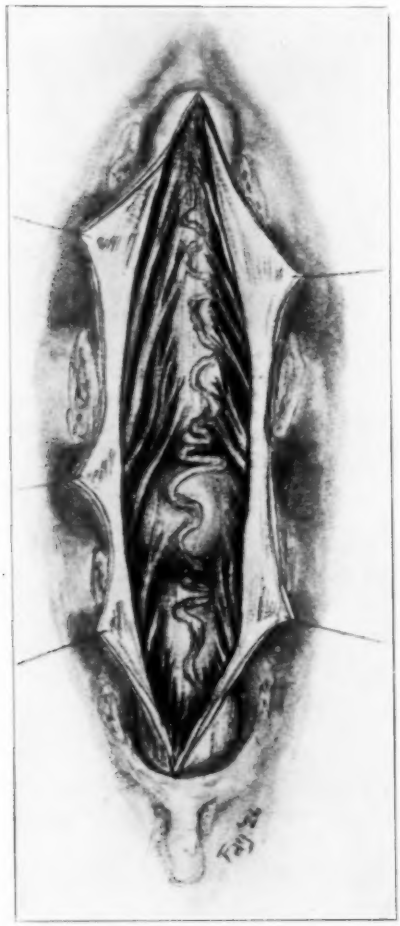

Fig. 39 (case 13).—Traumatic myelitis and swelling of the cord seen at operation sixteen hours after the injury. A fractured portion of the eleventh thoracic lamina was found pressing deeply on the cord.

THE FUNCTIONS OF THE CEREBELLAR NUCLEI AS DETERMINED BY FARADIC STIMULATION *

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For the investigation of the functions of the cerebellum, the method of faradic stimulation has been utilized much less extensively than the method of extirpation, partial or complete; in this respect the cerebellum differs greatly from the cerebrum, knowledge of the activities of which has been gained rather by electrical stimulations than by ablations. The advantages of faradic stimulation are indeed clearly apparent, since it serves to evoke activities which simulate closely normal living processes; on the other hand, extirpations, which subtract elements of these processes, leave a residue of morbid phenomena, partly of deficiency, partly of compensation, from which it is difficult to reconstruct the faculties of the parts removed.

The reason for the comparative neglect of the method of stimulation in the case of the cerebellum is to be found in the difficulty experienced in evoking reactions from the cortex; this is admitted by all observers [1] who found themselves compelled to use currents of great intensity; thus, many fallacies entered into their results, since the current frequently spread beyond the cerebellum to various tracts and nuclei, more particularly to the nucleus and rootlets of the accessorius nerve.

The serious discrepancies between the results of stimulation observed by different workers, together with the great intensities of current which they frequently employed, induced Horsley and Clarke [2] to deny the excitability of the cerebellar cortex and to ascribe the effects occasionally yielded by cortical stimulation to current diffusion to the cerebellar nuclei.

If we seek now for the reason for the failure to evoke reactions from the cerebellar cortex, we shall find it in the circumstance that the cerebellum governs not ordinary muscular movement but postural tone

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* Read before the Association for Research in Nervous and Mental Disease, New York, Dec. 29, 1926.

1. Miller, F. R.: The Physiology of the Cerebellum, Physiol. Rev. **6**:124, 1926.

2. Horsley, Victor; and Clarke, R. H.: The Structure and Functions of the Cerebellum Examined by a New Method, Brain **31**:45, 1908.

instead; this conception of cerebellar function, first suggested in a paper by Walshe,[3] is widely accepted at the present time, in spite of certain objections recently made to it, which will receive attention in later sections of this paper. Admitting, then, that the cerebellum is a mechanism of tonus, changes in tonus should be expected in consequence of stimulating the cerebellum; but these are not readily shown in the usual circumstances of an experiment; it is indeed only in the decerebrate condition that we may reasonably hope to induce changes in tonus, which might manifest themselves against the background of decerebrate rigidity.

The theory that the cerebellar cortex is inexcitable cannot, however, be longer maintained; in disproving it, Banting and one of us (F. R. M.) employed a reaction elicitable from the front of the cortex in the decerebrate animal;[4] it consists in inhibition of the decerebrate tonus and was originally described by Sherrington.[5] This reaction, we were able to show, is an expression of a state of excitation in the cortical neurons induced by faradization, thus showing that the cortex may be excited by this form of stimulation.

As the cerebellar cortex has been shown to be excitable, we may logically proceed to apply the same method of investigation to the cerebellar nuclei, since they are connected with the cortex and form important stations through which impulses travel from the various cortical areas to the centers of the midbrain and the medulla oblongata; they have their origin in the mantle layer of those portions of the metencephalic alar laminae that are destined to form the cerebellum.[6]

In the human cerebellum, in which the nuclei attain their greatest development and differentiation, their form and relationships have been studied extensively: following the usual description, from the outer side proceeding medialward, we find the nucleus dentatus, nucleus emboliformis, nucleus globosus and nucleus fastigii or roof nucleus. The nucleus dentatus is the most characteristic; its gray substance is wavy in outline and is folded so as to enclose a cavity, from the mouth of which the fibers stream forward which it contributes to the brachium conjunctivum.

This complexity of form is not encountered in simpler mammals; for instance, in the cat, the subject of the present investigation, the

3. Walshe, F. M. R.: On Disorders of Movement Resulting from Loss of Postural Tone, with Special Reference to Cerebellar Ataxy, Brain **44**:539, 1921.

4. Miller, F. R., and Banting, F. G.: Observations on Cerebellar Stimulations, Am. J. Physiol. **59**:478, 1922; read, December, 1921; Brain **45**:104, 1922.

5. Sherrington, C. S.: Decerebrate Rigidity and Reflex Co-Ordination of Movements, J. Physiol. **22**:319, 1897-1898.

6. Ranson, S. W.: The Anatomy of the Nervous System, Philadelphia, W. B. Saunders Company, 1921.

studies of Cajal [7] and Weidenreich [8] show that the nucleus dentatus is no more than a small extension of the central gray substance; it is compact in texture and its outer margin is slightly lobulated. The other nuclei show much less individuality than in man. Still less differentiation of the several nuclei is found in the lower mammals, such as the guinea-pig and the mole, in which the central gray mass shows only faint indications of a division into separate nuclei. Thus, the several nuclei arose in one mass and, although in man the nuclear units have much independence, even here slight bonds of gray matter exist between them.

Weidenreich's researches on the morphology of the cerebellar nuclei extended over the mammalian series and led him to adopt, in respect to the nuclei, designations different from the conventional terms of human anatomy; thus he refers to the nucleus dentatus as the nucleus lateralis, to the nucleus emboliformis as the nucleus lateralis anterior, to the nucleus globosus as the nucleus lateralis posterior and to the nucleus fastigii as the nucleus medialis. While these terms have obvious advantages in emphasizing the positions and relationships of the nuclei, the names used in human anatomy have been extensively employed by many authors when referring to the nuclei of lower mammals. They are used, for example, by Winkler and Potter [9] in their atlas of the cat's brain and by Allen [10] in a recent paper dealing with the distribution of the cerebellofugal fibers. It is true that the author mentioned last makes a separation from the point of view of function, there being two distinct nuclei: the median of these is the nucleus fastigii, while the lateral comprises the nucleus dentatus and the nucleus interpositus, which is composed of the nucleus emboliformis and the nucleus globosus; but throughout his paper, Allen retains the older nomenclature. This we have done ourselves and have adhered to the names used in human anatomy.

The neurons composing the cerebellar nuclei, in appearance like those of the inferior olive,[7] represent important stations in the course of the cerebellar reflex arcs, linking, as they do, the cerebellar cortex to various prespinal centers. The axons of the cortical Purkinje cells form elaborate synapses around the cells of the nuclei.[7] As to which cortical areas are connected to the several nuclei, we have enlighten-

7. Cajal, S. R.: Histologie du système nerveux, Paris, A. Maloine, 1911, vol. 2.

8. Weidenreich, F.: Zur Anatomie der centralen Kleinhirnkerne der Säuger, Ztschr. f. Morph. u. Anthrop. **1**:259, 1899.

9. Winkler, C., and Potter, A.: An Anatomical Guide to Experimental Researches on the Cat's Brain, Amsterdam, W. Versluys, 1914.

10. Allen, W. F.: Distribution of the Fibres Originating from the Different Basal Cerebellar Nuclei, J. Comp. Neurol. **36**:399, 1923-1924.

ment from the researches of Horsley and Clarke [11] and of Allen;[10] we may summarize their more important conclusions, expressing them, as far as possible, in relation to Ingvar's newer method of subdividing the cerebellum.[12]

The nucleus fastigii receives fibers mainly from the median cortex ("vermis") and also from most other cortical areas; the nucleus globosus is in relation to the median cortex and not to the lateral cortex ("hemisphere"); the nucleus dentatus is connected with the lateral cortex (hemisphere) and with the median cortex as represented by the lobus medius (Ingvar), a region formed by the declive and tuber. The nucleus fastigii receives further direct and indirect vestibulocerebellar fibers and possibly also spinocerebellar fibers [10] (Villiger and Addison [13]).

We may now sketch briefly the further course of the cerebellar reflex arcs, dealing with them only so far as they are concerned in our experimental reactions. Axons from the nucleus dentatus, together with others from the nuclei emboliformis and globosus, pass forward in the brachium conjunctivum. These fibers cross the middle line in Wernekink's commissure and, after giving off a descending tract destined for the reticular formation of the pons and the medulla oblongata, come into close association with the cells of the nucleus ruber. In the view of Hatschek [14] and Marburg,[15] the orally situated, small-celled component of this nucleus (nucleus ruber parvicellulatus) receives fibers from the dentate nucleus, while the aboral, large-celled component (nucleus magnicellulatus) receives fibers from the emboliform and globose nuclei. Whether we may apply these statements unreservedly to the red nucleus of the cat is not certain, and various facts make us skeptical on this point. It is true that the caudal part of the nucleus of this animal contains a preponderance of large or giant cells and the oral part a preponderance of small cells (Hatschek [14] and Monakow [16]), but it is not possible to divide the nucleus strictly into a large-celled and a small-celled part.

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11. Clarke, R. H., and Horsley, V.: On the Intrinsic Fibres of the Cerebellum, its Nuclei and its Efferent Tracts, Brain **28**:13, 1905.

12. Ingvar, S.: Zur Phylo- und Ontogenese des Kleinhirns, Folia Neuro-Biol. **11**:205, 1918.

13. Villiger, E., and Addison, W. H. F.: Brain and Spinal Cord, Philadelphia, J. B. Lippincott Co., 1925.

14. Hatschek, R.: Zur vergleichenden Anatomie des Nucleus Ruber Tegmenti, Arb. a. d. Wiener Neurol. Inst. **15**:89, 1907.

15. Marburg, O.: Handbuch der Neurologie des Ohres, vol. 1, Berlin and Vienna, Urban und Schwarzenberg, 1923.

16. Von Monakow, C.: Der rote Kern, die Haube, usw., Arb. a. d. Hirnanat. Inst. in Zürich, nos. 3 and 4, 1909-1910.

Allen finds that the emboliform and globose fibers in the guinea-pig end in the large-celled part of the red nucleus, but the dentate fibers end there also, as well as in the small-celled part.[17] Mussen [18] has recently made a careful study of this question in the cat; by employing the stereotaxic instrument of Horsley and Clarke,[2] he was enabled to make small lesions, avoiding damage to contiguous parts; his results so far do not confirm those of Hatschek. We must be content, therefore, to regard the relations in this portion of the reflex arcs as unsettled at the present time. Undoubtedly, the reactions we have obtained from the lateral cerebellar nuclei employ the paths we have just outlined; their oral extensions to the thalamus and cerebral cortex we leave unnoticed, since they are not involved in the reactions of an animal decerebrated in front of the midbrain.

The principal descending tract from the red nucleus is the rubrospinal, its origin being in the nucleus magnicellulatus; after crossing the midline in the decussation of Forel, it extends downward, giving off collaterals around the ventral motor cells of the spinal cord; it doubtless forms a further part of the conducting path for our reactions from the lateral cerebellar nuclei. The other pathway, probably of less importance, is the rubroreticular, originating in the nucleus parvicellulatus and proceeding to the reticular formation of the pons, beyond which the reticulospinal tract conveys impulses downward to the cord. Mussen finds that damage to the nucleus parvicellulatus results in degeneration as far as the cervical cord.

The fastigiobulbar tract (the fasciculus uncinatus of Russell,[19] faisceau en crochet or faisceau cérébello-bulbaire of van Gehuchten [20]) is formed by fibers from the nuclei fastigii of both sides, those from the opposite nucleus crossing the midline (Kappers [21] [p. 735, fig. 400] and Marburg [15] [p. 323]); it loops around the brachium conjunctivum, passing medially to the corpus restiforme and is distributed finally to the reticular formation of the pons and medulla oblongata (van Gehuchten [20] and Edinger [22]). According to an opinion expressed to us personally by Prof. W. H. F. Addison, we may regard this tract as subserving, in large measure, the responses which we have obtained from the nucleus fastigii. Tracts originating in the roof nuclei of both

17. Allen, W. F.: Personal communication to the authors.

18. Mussen, A. T.: Personal communication to the authors.

19. Russell, J. S. R.: Degenerations Consequent on Experimental Lesions of the Cerebellum, Phil. Trans. Roy Soc. **186**:633, 1896.

20. Van Gehuchten, A.: Le faisceau en crochet de Russell ou faisceau cérébello-bulbaire, Le Névraxe **7**:119, 1905.

21. Kappers, C. U. A.: Die vergleichende Anatomie des Nervensystems, ii Absch., Haarlem, De Erven F. Bohn, 1921.

22. Edinger, L.: Vorlesungen über den Bau der nerv. Zentralorgane, Leipzig, F. C. W. Vogel, 1911, vol. 1.

sides and passing via the restiform body are possibly concerned also; some of these terminate in the nucleus of Deiters; others pass to the tegmentum of the opposite side as ventral external arcuate fibers. The reticulospinal and vestibulospinal tracts form the final links downward to the spinal centers.

The only previous work on stimulation of the cerebellar nuclei that has come to our notice is that of Horsley and Clarke;[2] their results are mentioned only incidentally in the course of a long paper dealing chiefly with their stereotaxic instrument. They found that faradic stimulation of the nucleus dentatus yielded flexion of the elbow of the same side, while galvanic stimulation of this nucleus yielded extension of the joint, a response also obtained to faradism when the nucleus was fatigued. Stimulation of the nucleus fastigii yielded conjugate deviation of the eyes. Our own results, which we shall now describe in detail, have already been recorded.[23]

METHODS

The technical details of our experiments are as follows: The cat is anesthetized with chloroform and ether; a cannula is tied into the trachea and both carotid arteries are ligated. The nervus accessorius is then isolated on the side of the stimulation (usually the right) and cut just above its entry into the cleidomastoid muscle; the purpose of this procedure is to avoid vitiation of results by escape of the current to the nerve, a matter often ignored by earlier workers. In reality the operation is usually unnecessary, since responses may be excited in the cerebellar nuclei with currents much too weak to cause effects of escape.

The animal is next laid on the operating table in the ventral decubitus with a warm water-bottle placed transversely so as to support the abdomen just in front of the hindlegs; the head is secured in a Czermak head-holder, adjusted so that the line of the mouth is inclined downward, making an angle of 45 degrees below the horizontal; the reason for this will be explained later. The skull is trephined on each side in the parietotemporal region, and the bone between the two openings is removed with rongeur forceps; the superior sagittal sinus is ligated in front and behind and the intervening part removed; the dura is then opened up extensively. The frontal lobes of the cerebral hemispheres are first removed and then the occipital poles raised and pressed forward; in this way the colliculi become visible. The brain stem is then transected by a cut which passes dorsally just in front of the superior colliculi and ventrally in front of the infundibulum; the remainder of the hemispheres is then removed, the caudal and basal parts sometimes being left. The lateral geniculate bodies are usually removed, the medial sometimes retained. During the removal of the brain, pressure is maintained by the finger and thumb of the assistant over the vertebral arteries just behind the wings of the atlas, after the manner of Sherrington; our operation is, in fact, a modification of his original method of decerebration, whereby the caudal parts of the thalami are retained and the nuclei rubres thus preserved intact, a condition essential for our cerebellar stimulations. Following the decerebration, the base of the skull is packed tightly with absorbent cotton and the skin of the head closed over the opening. It happens, not infrequently, that movements of

23. Miller, F. R., and Laughton, N. B.: Nature **118**:516, 1926.

progression appear, sometimes of such violence as greatly to interfere with the continuance of the experiment; in these cases we usually make a second transection slightly more caudal than the first and thereby eliminate the movements. Sometimes we employ an intraperitoneal injection of urethane. Although there is some chance of impinging on the red nucleus with the second cut, we usually have more success with this procedure than with the use of urethane. All anesthetics seem to interfere seriously with the cerebellar reactions.

After an interval sufficient for the recovery from shock is allowed, the muscles are detached from the occipital bone on the right side and the bone itself is chipped away, permitting the exposure of the cerebellum. To expose the cerebellar nuclei, a horizontal cut is made on a level with the dorsal surfaces of the inferior colliculi, the head being in the position already mentioned; a cut of this kind approaches the nuclei closely; the cerebellar tissue above this section is then excised, but in doing so it is desirable to avoid removal of the rostral parts of the cerebellar cortex, the ablation of which causes intense rigidity in the foreleg, a symptom which often masks the effects of nuclear stimulation.

There is one matter that we must leave largely unexplained for the present, and that is that considerable rigidity develops from our transection of the neuraxis; this observation is at variance with that of Rademaker,[24] who claims that the tonus is normal until the large-celled red nucleus is impinged on by the transection. This cannot be the case in most of our experiments, since our section should miss the nucleus or at any rate its large-celled part, to judge from figure 219 in Magnus'[25] work on body posture. However, other workers (Warner and Olmsted[26]) have reported an increase of extensor tone on removal of the frontal lobe, an observation agreeing with the statements of Sherrington[5] and Weed[27] as to the presence of a cortical descending tract inhibitory of tonus. Whether the increased tonus in our experiments is of exactly the same kind as that studied by Rademaker we cannot say, but it resembles that described by the other investigators; in any case, it is of considerable importance in our cerebellar reactions.

In order to observe the cerebellar reactions, the animal is suspended freely above the table; to do this, a string is passed through the skin of the back near the tail. The animal is raised by this and by the head-holder, which is then elevated so that the position is horizontal; meantime, the adjustment of the head-holder is preserved, the line of the mouth making an angle of 45 degrees below the horizontal: this is Magnus' position for minimal tonus in the extremities from labyrinthine stimulation.[25] Our reason for adopting this position is that the nuclear reactions appear in purer form when the rigidity is reduced as far as possible. We observe mainly the effects on the limbs, trunk and tail, paying some

24. Rademaker, G. G. J.: Die Bedeutung der roten Kerne, usw., Berlin, Julius Springer, 1926.

25. Magnus, R.: Körperstellung, Berlin, Julius Springer, 1924.

26. Warner, W. P., and Olmsted, J. M. D.: The Influence of the Cerebrum and Cerebellum on Extensor Rigidity, Brain **46**:189, 1923.

27. Weed, L. H.: Observations upon Decerebrate Rigidity, J. Physiol. **48**:205, 1914.

attention also to ocular changes. It is clear that movements of the neck cannot be observed; indeed, it is not feasible accurately to localize in the cerebellum if the head is free to move. The indifferent electrode, covered by a pad soaked in saline solution, is applied to the clipped skin in the lower lumbar region. For stimulation a Cambridge inductorium is used; it is operated by three Edison storage cells, an ammeter and rheostat being included in the circuit. In a few of the earlier experiments coils of other makes were employed.

OBSERVATIONS

Following the operative procedures mentioned, the animal being suspended in the way just described, the forelimb on the side of the cerebellar ablation customarily assumes a characteristic posture: it is flexed so that the elbow forms a right angle, with the paw advanced forward; we have called this the "hand-shake position," since it reminds one of a trained cat giving its paw. It is probably of the same nature as the "military salute" of Rynberk,[28] although to produce this only the crus I was removed, whereas we remove both crura I and II. If the limb is extended and then released it returns, in a curious leisurely way, to the hand-shake position, a reaction doubtless of the same nature as the anisothenia of André-Thomas and Durupt:[29] here following ablation of a given cerebellar region, the limb shows a deviation from the normal, the consequence of a disordered distribution of tonus.

In cases in which the foreleg is only slightly flexed as a result of the cerebellar removal, stimulation of the limb itself, especially squeezing the paw, elicits further flexion at the elbow, the "hand-shake reflex," as we term it; this type of forelimb flexion is carried out in a peculiar retarded fashion in comparison with such a reflex as the flexion reflex of the hindleg of the spinal cat; it reminds one of a slow motion picture of a reflex movement. The hand-shake reflex is sometimes highly excitable and may be evoked by stimulation of the neck, head and other parts of the body as well as of the limb itself. It may frequently be elicited as an auditory reflex by whistling into either ear, the stimulation from the opposite side being the more potent.[30] In some experiments, the flexion movement may be repeated several times as though the limb were performing movements of progression.

We have found the experimental conditions just described the most favorable for stimulation of the cerebellar nuclei. The position of the particular nucleus having been determined approximately by measurements, the cut section of cerebellum in that neighborhood is explored

28. Van Rynberk, G.: Das Lokalisationsproblem im Kleinhirn, Ergebn. d. Physiol., 1908, p. 653.

29. André-Thomas and Durupt, A.: Localisations cérébelleuses, Paris, Vigot Frères, 1914.

30. Miller, F. R.; Laughton, N. B., and Waud, R. A.: Reflexes in the Decerebrate Cat After Cerebellar Ablations, Tr. Roy. Soc. Canada, 1926, vol. 20, sec. v, p. 241.

carefully with a current of minimal intensity applied by a Sherrington unipolar electrode; we are here guided largely by the appearance of a definite reaction.

Our experience has shown us that the reaction most characteristic for the lateral nuclei is a further flexion of the ipsilateral foreleg; we seek then to induce this reaction, or some other of a definite type and, having found it, reduce the current still further and determine the area yielding it; this it usually extensive, but it is strictly limited in certain directions, particularly laterally, indicating that the response is evoked from definite structures. We have often found the response to be yielded both by the nucleus and by fibers that evidently proceed from it; but, as the nucleus can be identified more positively in the sections made later than nerve fibers, the origin of which may not be apparent, we consider localization satisfactory only when it is obtained from the nucleus itself. Having ascertained, then, the responsive area, it is outlined by inserting fine horse-hair bristles; the focus or point of greatest intensity is also marked in the same way; sometimes the focus alone is marked, although if conditions permit, we find it best to outline the area as just stated; the positions of the bristles are indicated on a sketch made at the time of the experiment.

After the completion of the experiment, the brain is fixed in a solution of formaldehyde and photographed from the dorsal and ventral sides, so as to show both the positions of the bristles and also the level of decerebration; outline drawings are also made to serve as guides to the photographs. Ultimately, serial sections are cut in celloidin through the bristles, and the sections are stained by the Weigert hematoxylin method. We have frequently found that, in the case of the reaction mentioned (flexion of the ipsilateral forelimb), the structure marked is the nucleus emboliformis. The following protocol is an instance of this.

Experiment of Feb. 26, 1925.—After the removal of the right lobulus anisformis, the condition of the animal was as follows: The right foreleg was in the "hand-shake position"; it was plastic and adducted, with the claws extended; further flexion was readily evoked by stimulation of the limb or by auditory stimuli. The left foreleg was in rigid extension. The hindlegs were in moderate extensor rigidity, which was less intense in the right. Unipolar faradization of the right nucleus emboliformis, with a weak current, the secondary distance being 290 to 230 mm., yielded: Right foreleg, flexed more and drawn caudally, adducted, paw, palmar flexed; left foreleg, rigidity decreased; right hindleg, flexed; left hindleg, unchanged. The body curved with the concavity on the side of stimulation. After stimulation the limbs and body returned to the condition that existed before stimulation.

Examination of the brain showed that transection was just in front of the superior colliculi dorsally and through the front of the infundibulum ventrally; a portion of the medial geniculate body remained on the left side, that on the right being completely removed (fig. 1). Weigert sections showed that the ablation

had been made exactly to the dorsal surface of the nucleus emboliformis and that the electrode had been applied directly to that surface of the nucleus. The bristle was inserted in the more caudal part of the nucleus; but the same effects were obtained just around the bristle and for about 2 mm. rostral from it; the reason that effects were maximal at the bristle was that there the nucleus was most completely exposed (fig. 2).

In calling this part of the nucleus the nucleus emboliformis, we are following the charts in Winkler and Potter's atlas; since the part stimulated was caudal, we might call it the nucleus lateralis posterior, follow-

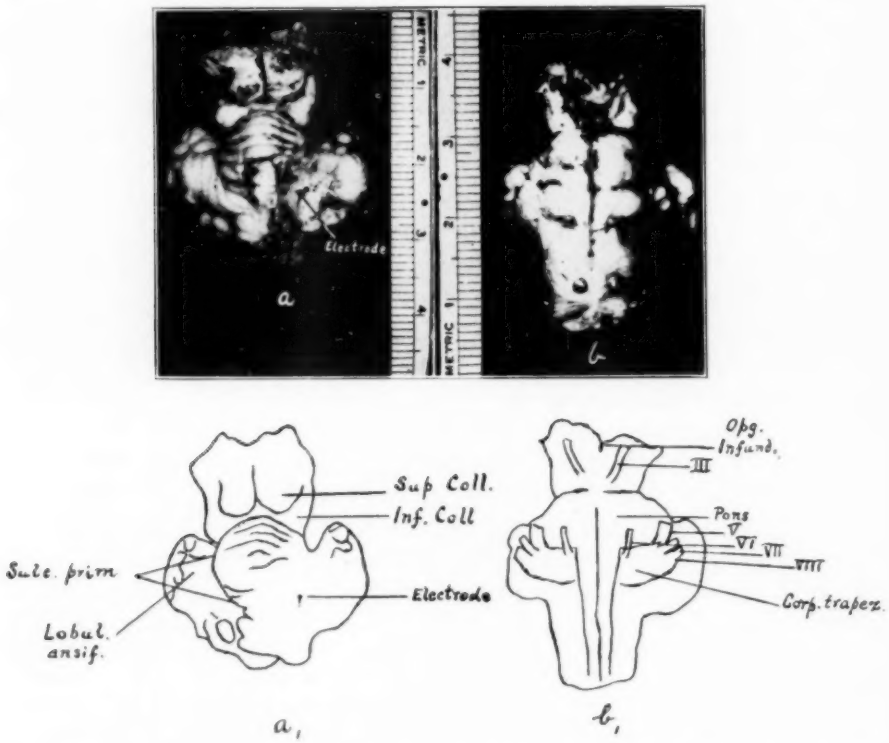


Fig. 1.—Appearance of brain in the experiment of Feb. 26, 1925, to show level of decerebration and position of electrode. *a* and a_1 are dorsal views, *b* and b_1 ventral views. *Corp. trapez.* indicates corpus trapezoideum; *Inf. coll.*, inferior colliculi; *Lobul. ansif.*, lobulus ansiformis; *Opg. Infund.*, opening of infundibulum; *Sulc. prim.*, sulcus primarius; *Sup. Coll.*, superior colliculi.

ing Weidenreich; on this basis, it would be the nucleus globosus; if so, it is clear that the response was the same from the nucleus emboliformis in front; stimulation thus reveals no difference between the oral and the caudal parts of the nucleus.

Thus we see that stimulation of the nucleus emboliformis or the nucleus globosus causes flexion of the ipsilateral foreleg as well as the

ipsilateral hindleg together with inhibition of the extensor rigidity in the contralateral foreleg, a reaction which might have been flexion if the limb had not already been stiffly extended; also, the body was rendered concave on the right side, clearly by an increase of tonus in the ipsilateral muscles. The nuclear stimulation obviously yields augmentation of the tonus of a large number of muscle groups with, undoubtedly, reciprocal inhibition in their antagonists. Inhibitory processes are clearly manifest in the relaxation of the extensor rigidity in the contralateral foreleg. The nucleus emboliformis can thus produce coordinated alterations in tonus or, expressing it otherwise, can cause changes in postural tone, both augmentor and inhibitory in nature.

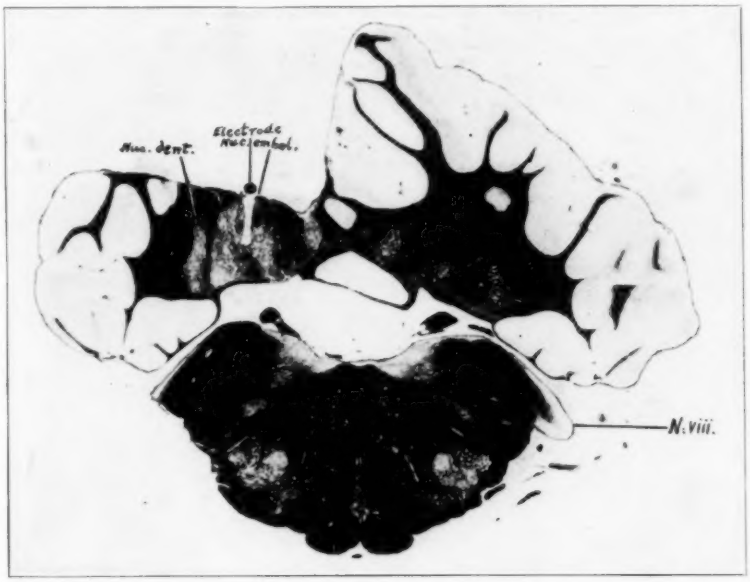


Fig. 2.—Frontal section (rostral surface) of cerebellum and medulla oblongata in experiment of Feb. 26, 1925, showing position of unipolar stimulating electrode over the right nucleus emboliformis; Weigert's hematoxylin stain. In this figure and in figures 3, 4, 6, 8, 10, 11, 13 and 15: *Nuc. dent.* indicates nucleus dentatus; *nuc. embol.*, nucleus emboliformis. The stab under the electrode was made by the bristle which marks the point stimulated on surface.

Another experiment in which the nucleus emboliformis was stimulated at a point somewhat more orally yielded similar results:

Experiment of July 20, 1923.—After removal of the right lobulus ansiformis, the condition of the animal was as follows: The right foreleg was in the handshake position and was plastic; the left foreleg was in rigid extension. The right hindleg was in rigid extension; the left was soft and plastic. Unipolar stimulation of the right nucleus emboliformis with a weak current and a secondary distance of 160 mm. yielded: Right foreleg, flexed further; paw, palmar flexed and

adducted; left foreleg, softer; hindlegs: right, softer and left, harder. The tail was raised, then rotated quickly in clockwise direction when looked at from the head end. A quick lateral nystagmus ceased, while the eyeballs were rotated counter-clockwise on their visual axes. After stimulation, the right foreleg showed extension rigidity as a "rebound" reaction; the left foreleg hardened; the left hindleg softened and the right hardened by rebound. The tail dropped slowly.

Sections showed that the electrode was applied above the lateral margin of the right nucleus emboliformis, over which a thin layer of white fibers remained; it seems probable that the medial part of the dentate nucleus was also stimulated in this experiment. Since the fibers intervening between the nuclei and the electrode are destined for the nuclei, stimulation of them doubtless revealed the action of the nuclei (fig. 3).

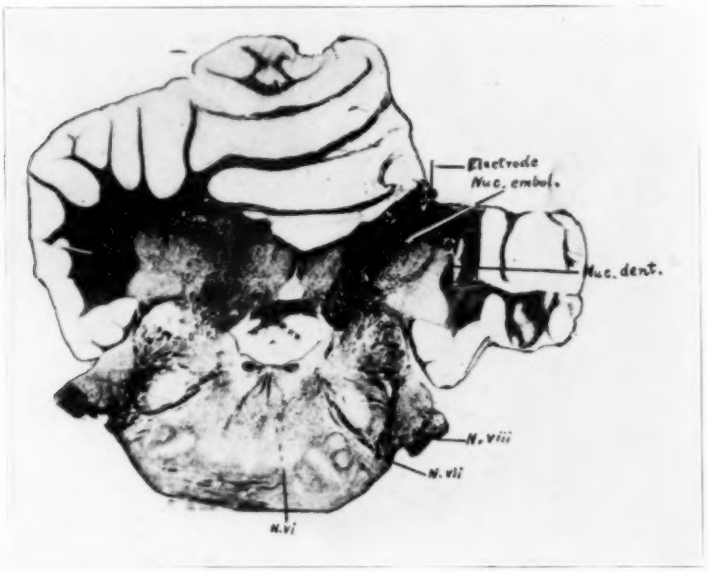


Fig. 3.—Section (caudal surface) showing position of unipolar electrode over the right nucleus emboliformis in experiment of July 20, 1923.

A variation of the responses of the forelimbs just described is encountered when both are in extensor rigidity; this condition develops in consequence of the more rostral parts of the cerebellum having been removed in making the ablation to expose the nuclei; these cerebellar regions are known to exert, when faradized, an inhibitory influence on decerebrate tonus. The type of nuclear response obtained under these conditions is illustrated by the following protocol:

Experiment of Oct. 11, 1922.—After decerebration with the Sherrington decerebrator, horizontal slices were removed from the cerebellum to the level of the nuclei; as a result, the rigidity was greatly increased in both forelegs. Faradization over the left nucleus emboliformis with a secondary distance of

176 mm. yielded inhibition of the tone and some flexion of the left foreleg with similar, though weaker, effects in the right foreleg. After stimulation there was a "rebound" increase of rigidity in both legs. These reactions are doubtless the same as those obtained in the experiment of Feb. 26, 1925, the movement of flexion in the ipsilateral foreleg being somewhat impeded by the decerebrate tonus present in it. Examination of sections proved that the electrode had been applied directly to the dorsal surface of the left nucleus emboliformis; this circumstance explained the refined character of the localization in this experiment (fig. 4).

An opposite result, as regards the ipsilateral foreleg, was obtained from the emboliform nucleus in the experiment of July 28, 1926; here the limb extended instead of flexing.

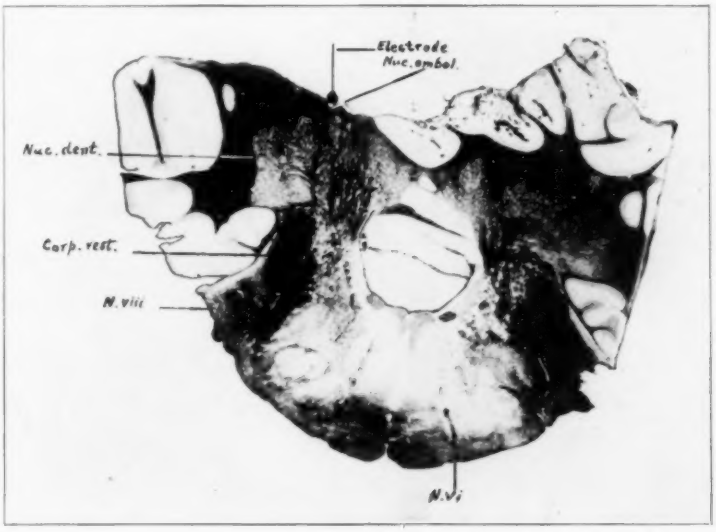


Fig. 4.—Section (caudal surface) showing position of unipolar electrode over the left nucleus emboliformis in experiment of Oct. 11, 1922. *Corp. rest.* indicates corpus restiforme.

After the right-sided cerebellar ablation, the condition was as follows: Right foreleg flexed at elbow; left foreleg in rigid extension; right hindleg partly flexed; left hindleg in rigid extension. Unipolar faradization of the right nucleus emboliformis with a secondary distance of 170 mm. yielded: Extension of right foreleg, the left usually being unchanged, though it was flexed once; hindlegs: right flexed and left extended. The base of the tail was turned to the left. After stimulation, the forelegs and tail returned to the condition before stimulation; the right hindleg was extended and the left flexed, there being plantar flexion of the paws of both limbs.

Examination of the brain showed that the plane of decerebration lay dorsally in front of the superior colliculi and ventrally in front of the mamillary bodies; both medial geniculate bodies were removed (fig. 5).

Sections showed that the electrode had been applied over the nucleus emboliformis, being separated from it by a thin layer of fibers (fig. 6).

The most constant and characteristic component of the responses elicitable from the nucleus emboliformis is, as already stated, the powerful flexion of the ipsilateral foreleg; this takes place best if the limb is already in moderate flexion; but it also takes place, though less obviously, if the limb is in extensor tone. The opposite result, namely, extension, reported in the foregoing, appears unquestionably to have been obtained from the nucleus, the level of stimulation lying, in fact, between those in two other experiments, both of which yielded flexion. It will be remembered that Horsley and Clarke obtained extension

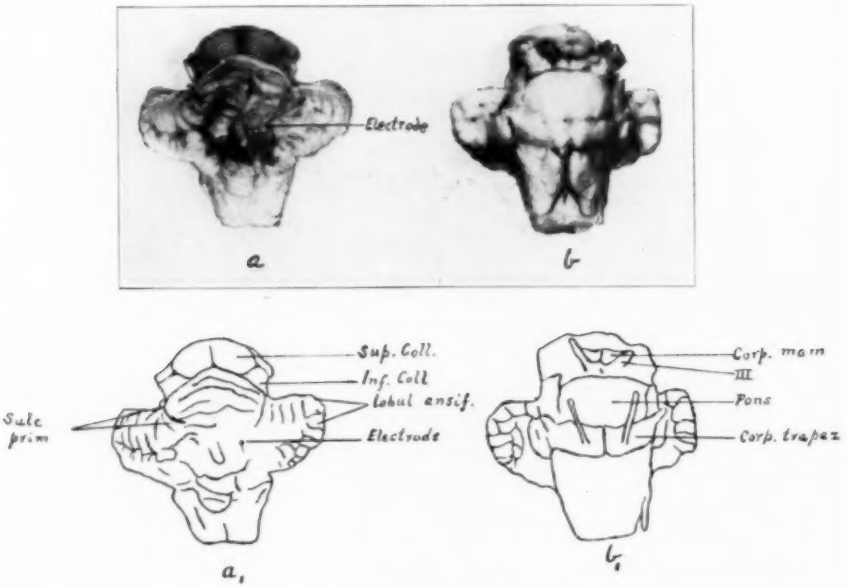


Fig. 5.—Brain in experiment of July 28, 1926. *Corp. mam.* indicates corpus mamillare.

instead of flexion when the nucleus was fatigued; some such factor may have entered into our own result; or possibly, a slight difference in position of the stimulation may have been responsible, as happens in the cerebral cortex.

Other experiments lead us to think that the action of the dentate nucleus is, in some degree, similar to that of the emboliform nucleus, a circumstance dependent on the close anatomic relationship of the two nuclei. The following protocol supports this statement:

Experiment of Nov. 26, 1925.—After removal of the right lobulus ansiformis, the condition of the animal was as follows: The right foreleg was in partial flexion, the forearm making an angle of 45 degrees with the vertical; stimulation of the limb itself or of the head, neck or different parts of the body evoked

repeated flexions like those of progression. The left foreleg was in moderate extension. Both hindlegs were rigid, showing some progression. Unipolar faradization of the outer part of the right nucleus dentatus with a secondary distance of 250 mm. yielded: Repeated flexions in the right foreleg like those seen in the reflexes; palmar flexion of the left forepaw; both hindlegs, more rigid; tail pointed to the left; body curved, concavity on left side. Following stimulation conditions became the same as before.

Examination of the brain showed that transection was dorsally in front of the superior colliculi and ventrally in front of the chiasma. The right medial geniculate body was removed; all but the caudal part of the left was removed (fig. 7).

Sections showed that a small portion had been removed from the outer side of the right nucleus dentatus and that stimulation had been applied to the nucleus

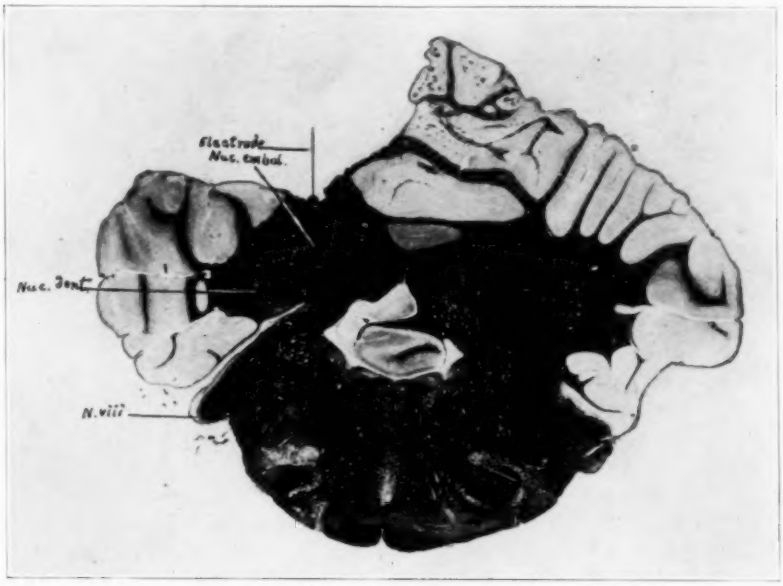


Fig. 6.—Section (rostral surface) showing position of unipolar electrode over the right nucleus emboliformis in experiment of July 28, 1926.

in that place. The same reactions were yielded by points immediately rostral to that just mentioned and, since the sections showed nerve fibers below the electrode there, we consider that these were efferent fibers going from the nucleus dentatus to the brachium conjunctivum (fig. 8).

An experiment in which bipolar stimulation was applied to the outer part of the dentate nucleus and in which the results yielded were similar to those in the last experiment was that of Oct. 12, 1923.

After removal of part of the right lobulus ansiformis, the animal presented the following symptoms: the right foreleg was less rigid than the left; the latter was in extensor rigidity. The hindlegs were in extensor rigidity that was less intense than in the forelegs. The tail was directed to the right and elevated

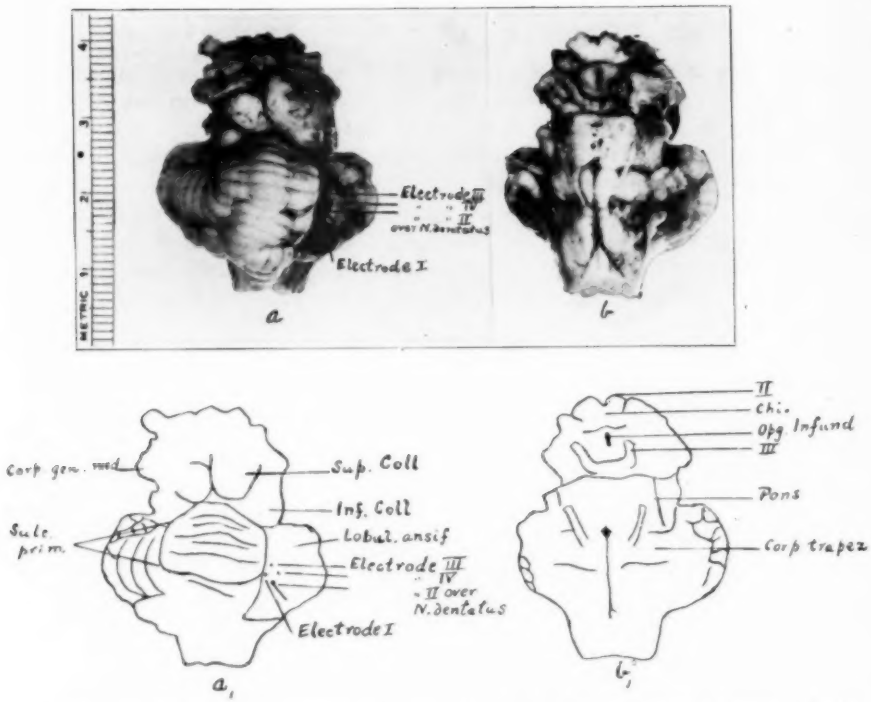


Fig. 7.—Brain in experiment of Nov. 26, 1925. *Chi.* indicates chiasma; *Corp. gen. med.*, corpus geniculatum mediale.

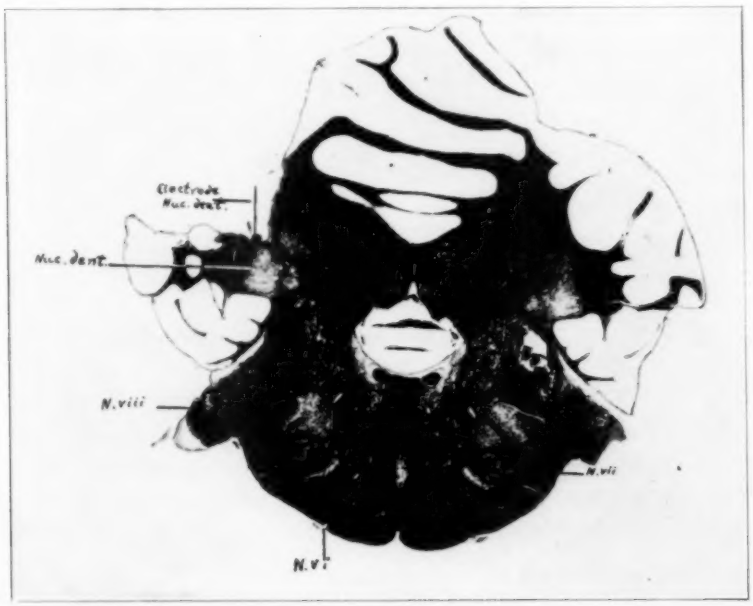


Fig. 8.—Section (rostral surface) showing position of unipolar electrode over the right nucleus dentatus (electrode II in figures 7*a* and a_1) in experiment of Nov. 26, 1925.

slightly. Bipolar stimulation of the right dentate nucleus, with one of the electrode poles in front and one behind and a secondary distance of 85 mm. (Stoelting coil), elicited the following changes: The right foreleg, flexed in hand-shake position, also adducted; the left, unchanged. The right hindleg was in rigid extension, with spreading of the claws; the left was slightly softer. After stimulation the forelegs were in extreme rigidity by extensor "rebound" and were also adducted, crossing each other; both hindlegs were in strong extensor rigidity. The tail was more elevated than before stimulation. It was observed in the course of this experiment that the application to the exposed cerebellar

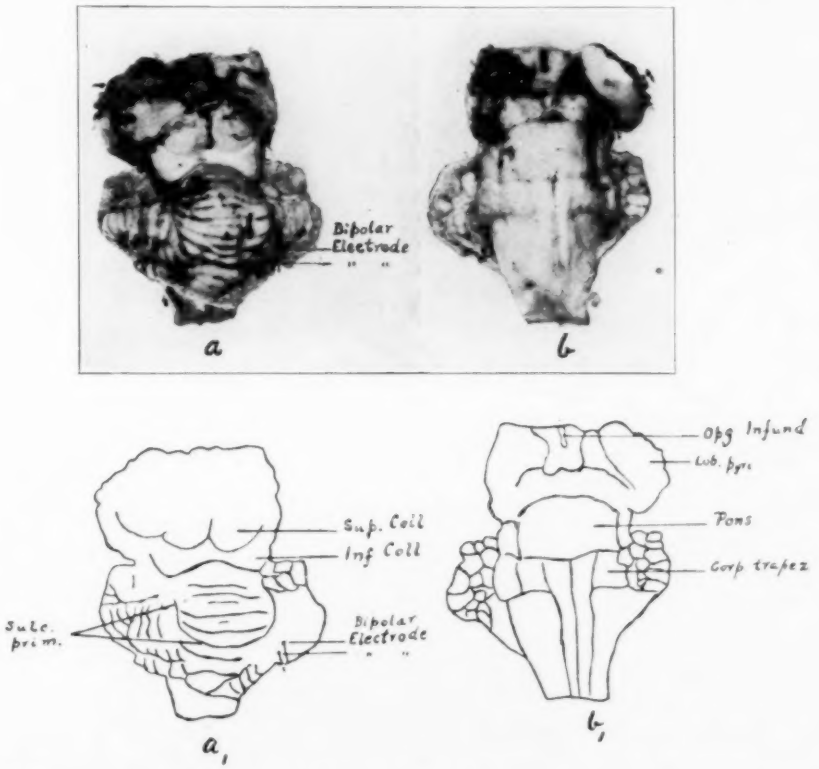


Fig. 9.—Brain in experiment of Oct. 12, 1923. *Lob. pyri.* indicates lobus pyriformis.

section of a pad soaked in warm saline solution caused a reaction in the right foreleg like that which occurs on faradization of the nucleus: the leg was adducted and assumed the hand-shake position; in this case the warm saline solution evidently stimulated the highly excitable cells of the nucleus.

Examination of the brain showed that the plane of decerebration lay dorsally in front of the superior colliculi, being slightly more rostral on the left side; ventrally, it was just in front of the opening of the infundibulum (fig. 9).

Sections showed that the front electrode of the pair had been close to the dorsolateral part of the dentate nucleus, being separated from it by a thin layer of fibers; the rear electrode was over the more caudal part of the same nucleus,

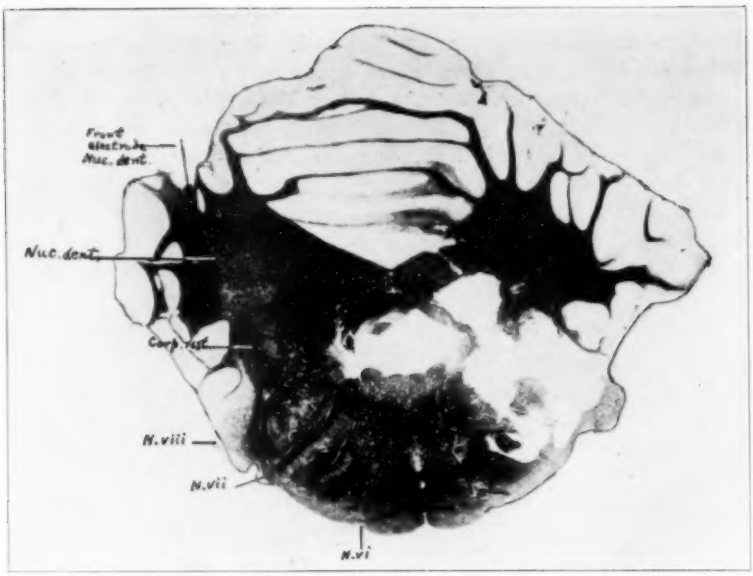


Fig. 10.—Section (rostral surface) showing position of front electrode of pair over the right nucleus dentatus in experment of Oct. 12, 1923.

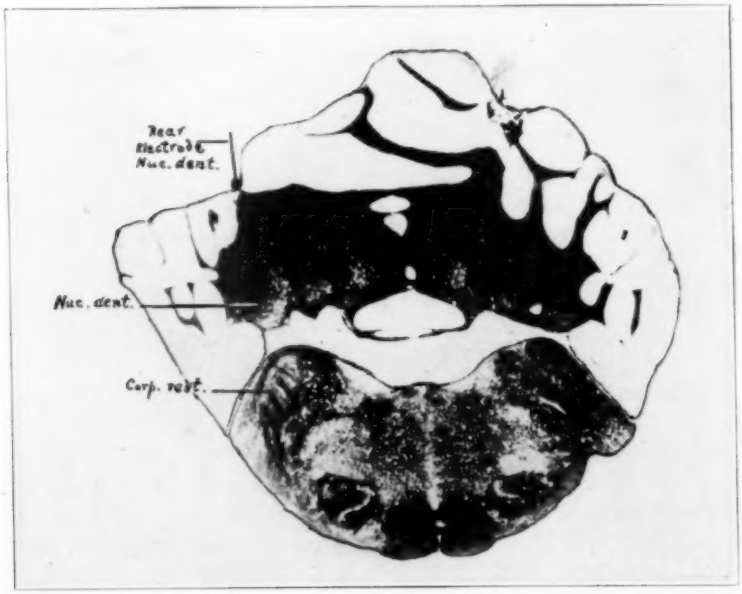


Fig. 11.—Section (rostral surface) showing position of rear electrode of pair over the right nucleus dentatus in experiment of Oct. 12, 1923.

though separated from it by a thicker layer of fibers. Since the fibers are mainly or entirely destined for the nucleus, it is clear that stimulation in this experiment revealed the action of the nucleus itself (figs. 10 and 11).

As in the case of the emboliform nucleus, the response from the dentate nucleus varies somewhat when the forelimbs are in extensor rigidity, owing to the type of cerebellar ablation. This is shown in the following experiment, in which the outer part of the nucleus dentatus was stimulated:

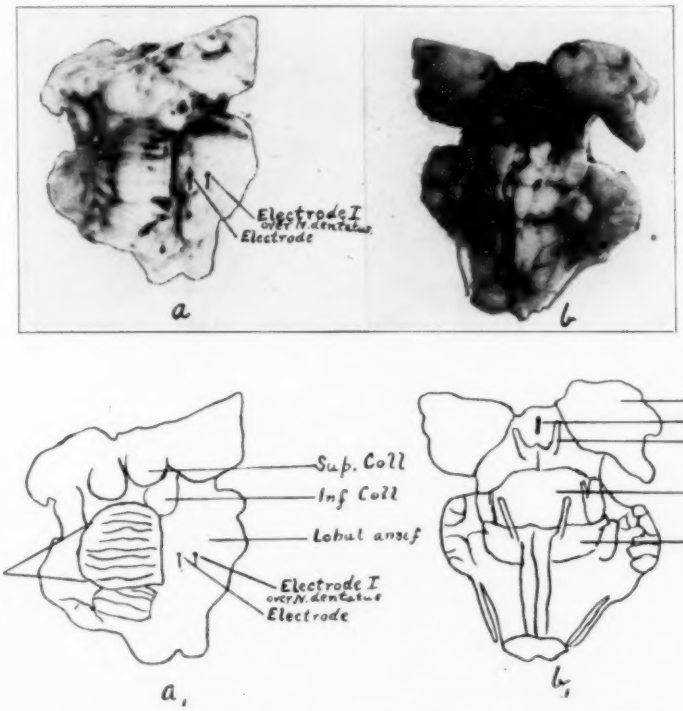


Fig. 12.—Brain in experiment of June 30, 1925.

Experiment of June 30, 1925.—The conditions existing after cerebellar ablation were: Both forelegs were in rigid extension, the right slightly more rigid. The hindlegs were in extension and were less rigid than the forelegs; the right was slightly more rigid. Unipolar faradization of the outer part of the right dentate nucleus, with a secondary distance of 120 mm., yielded: Inhibition of tone in the right foreleg so that it could be bent at the elbow; left foreleg, nothing definite; inhibition of tone in both hindlegs. Following stimulation, there were "rebound" augmentations of tone in the limbs. We are inclined to think that here also on stimulation an active flexion of the ipsilateral foreleg was prevented or masked by the extensor rigidity and was replaced by inhibition of tone.

Examination of the brain showed transection dorsally in front of the superior colliculi and ventrally in front of the opening of the infundibulum. Parts of the medial geniculate bodies and the pyriform lobes remained (fig. 12).

Sections showed that the electrode had been applied to the dorsolateral part of the right dentate nucleus (fig. 13).

In applying faradization to the nucleus fastigii, it was found advisable to make a horizontal cut with the knife all the way across the cerebellum, down to the dorsal surfaces of the nuclei on both sides.

Experiment of Dec. 10, 1925.—The conditions existing after cerebellar ablation were: Both forelegs were in the hand-shake position and were still further

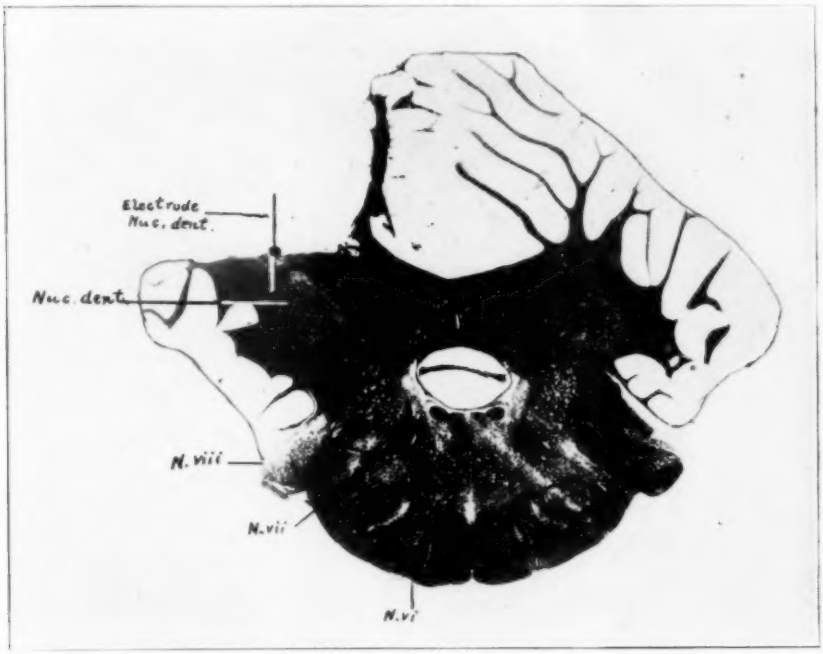


Fig. 13.—Section (rostral surface) showing position of unipolar electrode over the right nucleus dentatus (electrode I in figures 12*a* and a_1) in experiment of June 30, 1925.

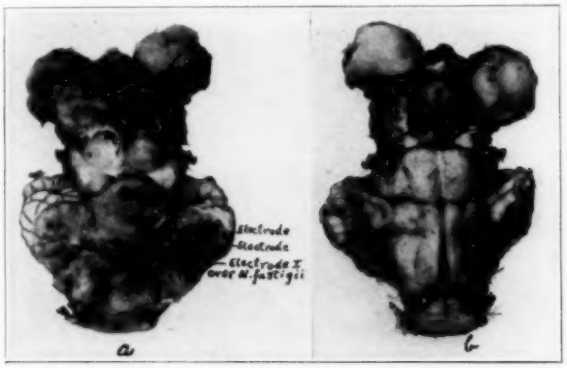
flexed on the slightest stimulation; the hindlegs showed some extensor rigidity with tendency to be flexed. Unipolar faradization over the right nucleus fastigii, with a secondary distance of 200 mm., yielded: strong flexion of both forelegs, with the toes slightly spread and the left foreleg slightly adducted; flexion in the right hindleg, with the toes spread apart; the left, unchanged. After stimulation, there was slow extensor "rebound" in both forelegs with adduction; the right hindleg returned to the state before stimulation; the left was unchanged.

Examination of the Brain.—The plane of transection dorsally was 3 mm. in front of the superior colliculi and ventrally through the front of the chiasma; parts of the pyriform lobes were left (fig. 14).

Sections showed that the electrode had been applied just above the lateral part of the right nucleus fastigii (fig. 15).

COMMENT

Pathways for Reactions from the Lateral Nuclei.—The efferent pathways employed by the reactions evoked from the lateral nuclei were already sketched in the introduction; they are the brachium conjunctivum, the nucleus ruber and the rubrospinal tract, and possibly also



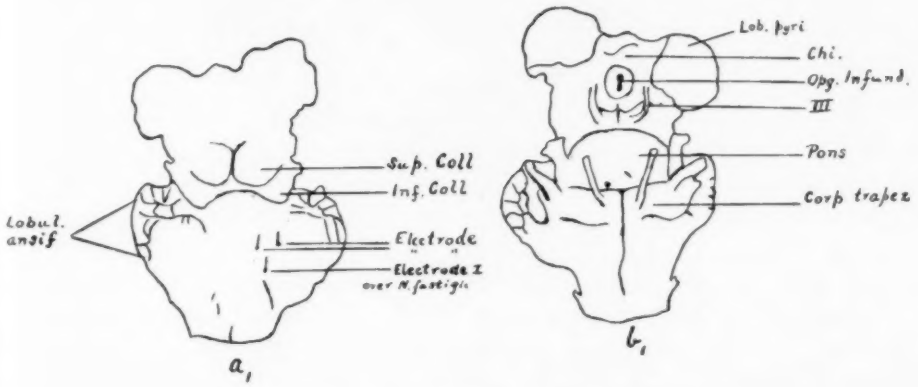


Fig. 14.—Brain in experiment of Dec. 10, 1925.

the rubroreticular tract. Impulses from the emboliform and globose nuclei pass mainly through the large-celled red nucleus and its extension, the rubrospinal tract; this is probably the route used in our most frequent reaction, that including flexion of the ipsilateral foreleg. The same or a closely similar route is probably used by impulses from the dentate nucleus. The course through the small-celled red nucleus and the rubroreticular tract may be traversed in other types of response; but of these, as of the more minute anatomic details within the red nucleus itself, we have as yet insufficient knowledge.

Pathways for Reactions from the Medial Nuclei.—The fastigiobulbar tract is, as already stated, probably the main pathway for the reactions evoked from the nucleus fastigii, reactions which have as an expressive feature flexion of both forelegs. To the explanation of this bilateral character of the response, the fastigiobulbar tract lends itself admirably, since it is formed by fibers issuing from the nuclei of both sides, those from the opposite side crossing the midline; its course and mode of termination have already been outlined. That this is the main route of impulses in our fastigial reactions seems certain, though the possibility of others also was suggested in the introduction.

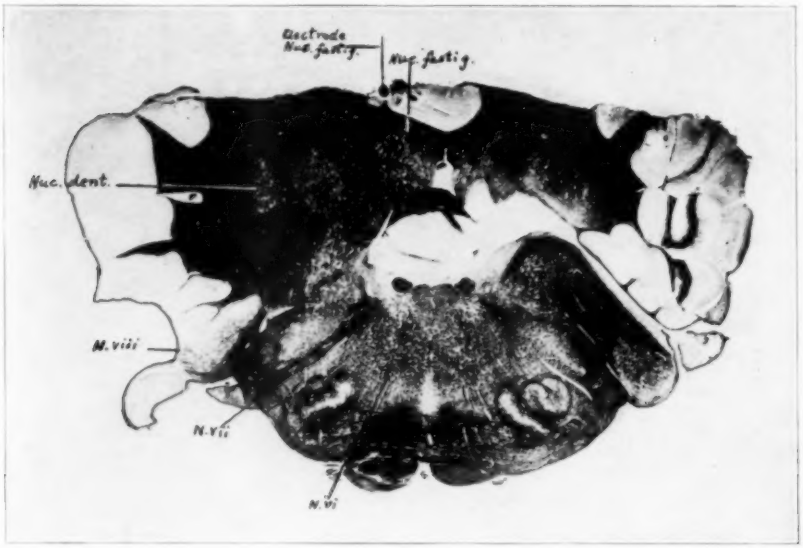


Fig. 15.—Section (rostral surface) showing position of unipolar electrode (electrode I in figures 14*a* and a_1) over the right nucleus fastigii in experiment of Dec. 10, 1925. *Nuc. fastig.* indicates nucleus fastigii.

Reactions of the Cerebellar Nuclei Regarded as Coordinated Changes in Postural Tone.—The reactions that we have obtained from the cerebellar nuclei are changes in postural tone, both augmentor and inhibitory in nature; taken with the inhibitory capacity of the rostral cerebellar cortex, they constitute the evidence for the view already proposed by one of us (F. R. M.)[1] that the cerebellar influence on tone is of both a positive and a negative kind. This is a radical modification and extension of Luciani's[31] doctrine of tonic action, which comes to be regarded as both augmentor and inhibitory in nature. Without

31. Luciani, L.: Human Physiology, London, The Macmillan Company, 1915, vol. 3.

referring to Luciani's sthenic and static actions, both of which are now considered as of minor importance in comparison with the tonic action, we must state that the (positive) tonic action of the cerebellum has been recently the object of criticism by Rademaker, as a result of his experience with cerebellar ablations. For an account of the elaborate investigations of this author, we are indebted to Prof. R. Magnus of the University of Utrecht [32] Briefly speaking, Rademaker rejects atonia or rather hypotonia as a symptom of cerebellar deficiency. He has developed a perfected technic for the complete extirpation of the cerebellum, using serial sections for purposes of control, until his experimental results were constant. Of a large series of dogs and cats operated on by his improved methods, a number continue in excellent health two years after the removal.

Immediately after the extirpation the animals suffer from shock, in which transitory "atonia" may appear; sometimes, on the other hand, rigidity or hypertonia is seen at this stage, which in young animals may last for eight days. In the succeeding period, in which the animals learn partially to compensate the disturbances of movement, one sees them creep with legs flexed, their bellies down, when on hard ground; but this is not due to atonia, since on grass or straw the animals run with strongly extended legs and, after a short time, learn to run upright on the stone floor as well. Either standing or running, they can carry sand-bags almost as heavy as themselves. Even with powerful pressure applied to the back of the standing animal, neither the forelegs nor the hindlegs give way. The neck and tail also show powerful tonus. In comparison with normal dogs and cats, the tonus is not diminished, and hence one may say that these animals do not show atonia.

We feel the greatest measure of confidence in the accuracy of these observations of Rademaker, both the technic of extirpation and the methods of examination of symptoms having reached a degree of refinement probably never before attained; we believe that the investigation has offered the most favorable conditions for the study of the symptomatology after cerebellar ablation. But the difficulty of reconstructing the functional properties of an organ after its removal has already been stressed; from the residue of morbid phenomena, partly of deficiency and partly of compensation, following removal of the cerebellum, it is well-nigh impossible to build up a complete theory of the functions of this organ. While, then, admitting in the fullest sense the great value of Rademaker's contributions, we may perhaps be allowed to interpret his observations in the light of our own conclusions based on stimulations.

32. Magnus, R.: Personal communication to the authors.

We have seen that in the early stage after extirpation Rademaker observed either atonia or hypertonia. We believe that this alternation of symptoms may be interpreted on the basis of our view that the cerebellar influence on tone is both augmentor and inhibitory; if we assume that these opposite cerebellar influences are of approximately equal potency, we may explain the predominance now of atonia, now of hypertonia, by slight differences in the mode of extirpation.

Coming now to the later stage of Rademaker, we may interpret the failure to observe atonia on our view of dual cerebellar control by the consideration that in the removal of the organ both the augmentor and inhibitory influences have been abolished; any possible inequality between them may well be equalized by various types of "compensation," the cerebral factors of which Luciani proved to be of such great importance.[31]

The positive and negative influences on postural tone are exerted presumably by different parts of the cerebellum or else by the same parts in changing functional states. Alterations of response of analogous kind are encountered in the cerebral cortex; here Sherrington and Leyton [33] found that the reaction of a point may vary, even to the extent of complete reversal, in consequence of previous stimulation of itself or of adjacent cortical points. These augmentor and inhibitory influences on tone we may consider as being predominantly over the ipsilateral muscles,[4] although it is probable that the influence of each part of the cerebellum may extend to most of the muscles of the body. For an excellent presentation of the modern doctrine of postural tone, we would refer to the recent review by Stanley Cobb.[34] And in spite of the objections of Rademaker, which we believe we have met satisfactorily, it is with postural tone that the cerebellum deals, in the positive and negative manner that we have outlined.

We have already pointed out in this paper that the tonus changes elicitable from the cerebellar nuclei are of a coordinated character, involving, no doubt, reciprocal innervation of antagonistic muscle groups. It would seem indeed that the postures assumed by the limbs, sometimes tonic flexion, sometimes tonic extension, are functionally equivalent to components of the tonic labyrinthine and tonic neck reflexes of Magnus and de Kleijn.[25] It is true that they have found these reflexes to persist after complete removal of the cerebellum, showing that the centers of coordination have a prespinal and spinal situation.

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33. Leyton, A. S. F., and Sherrington, C. S.: Observations on the Excitable Cortex of the Chimpanzee, Orang-Utan and Gorilla, Quart. J. Exper. Physiol. **11**:135, 1917.

34. Cobb, Stanley: Review on the Tonus of Skeletal Muscle, Physiol. Rev. **5**:518, 1925.

Nevertheless, the nuclear reactions suggest strongly that the cerebellum can influence the tonic reflexes and besides can evoke, by efferent impulses discharged through the nuclei, coordinated reactions like the reflexes or components of the reflexes. In the same way, efferent discharges from the cerebral cortex evoke in the subcortical centers coordinated activities closely simulating the reflex activities evokable in these centers by afferent nerves.

We thus consider the coordinated changes in tonus elicitable from the cerebellar nuclei of the cat as analogous to the coordinated movements elicitable from the cerebral cortex; only, while the latter are phasic, those from the cerebellar nuclei are tonic in character. The nuclear responses are, in our opinion, elementary tonic activities, which have been picked out by the analytical method of unipolar faradic stimulation. Responses of this kind may be synthesized and integrated, together with various reflex processes like those of Magnus and de Kleijn, so as to constitute the refined and delicate postural activities of the intact organism

SUMMARY

1. Faradic stimulation,[35] in most instances unipolar, was applied to the cerebellar nuclei in the decerebrate cat.

2. Characteristic responses of the nuclei are as follows: nucleus emboliformis and nucleus globosus: marked flexion of the ipsilateral foreleg; inhibition of decerebrate rigidity in the contralateral foreleg; flexion of the ipsilateral hindleg; curvature of the body; ocular movements; nucleus dentatus: flexion, sometimes repeated, of the ipsilateral

35. Since the present paper was read, an article by R. H. Clarke has been published posthumously (Experimental Stimulation of the Cerebellum, Brain **49**:557, 1926). In this article, the original statement of Horsley and Clarke[2] as to the inexcitability of the cerebellar cortex is reiterated; that this conclusion cannot be accepted has, however, been amply demonstrated.[4] Some of the responses of the limbs described by Clarke resemble those in our own experiments; they were attributed in the previous paper by Horsley and himself to actual stimulation of the dentate and other intracerebellar nuclei; but in this paper, Clarke states that the reactions were in reality caused by a spread of the current to Deiters nucleus and to various motor centers of the brain stem. So far as our own work is concerned we must assert positively that our reactions are produced by stimulation of the intracerebellar nuclei themselves and are not due, in any sense, to a spread of the current beyond the cerebellum. Phenomena caused by spread of the current are readily recognizable and were avoided by using currents of threshold value for the nuclei, the excitability of which was preserved by the technical methods of preparation. Some of our reactions are like those casually described by Horsley and Clarke[2] and attributed by them at the time to stimulation of the dentate nucleus. Our conclusions are thus more nearly in agreement with those of Horsley and Clarke than with those recorded in the posthumous paper of Clarke.

foreleg; palmar flexion in the contralateral foreleg; hindlegs more rigid; curvature of the body; nucleus fastigii: strong flexion of both forelegs; flexion of the ipsilateral hindleg.

3. Efferent pathways for reactions of the lateral nuclei are through the brachium conjunctivum, nucleus ruber and rubrospinal tract, possibly also the rubroreticular tract.

4. The principal efferent pathway for reactions of the nucleus fastigii is through the fastigiobulbar tract (fasciculus uncinatus of Russell).

5. The responses of the cerebellar nuclei are coordinated changes in postural tone, with augmentor and inhibitory components. They are regarded as analogous to the coordinated movements elicitable from the cerebral cortex; the cerebellar nuclear responses are tonic, and the cerebral responses, phasic.

RESORPTION OF CEREBROSPINAL FLUID THROUGH THE CHOROID PLEXUS

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The normal direction of flow of the cerebrospinal fluid has been indicated and its main points of exit from the subarachnoid space have been demonstrated by Weed [1] in 1914 and 1923, using careful physiologic controls. By the method of introducing isotonic potassium ferrocyanide and iron ammonium citrate solution into the subarachnoid space under pressures closely approaching the normal, he showed that the chief channels of escape of the cerebrospinal fluid were the arachnoid villi. If, however, a hypertonic solution of sodium chloride were previously injected into a vein, accessory paths of elimination were opened (probably due to a reversal in direction of flow) along the perivascular spaces and through the ependymal cells lining the ventricles, into the capillaries and venules of the brain substance.

Foley,[2] using Weed's method with modifications, substantiated these observations, but also showed the presence of Prussian blue granules within the vessels of the choroid plexuses, indicating a reversal of flow here, also. Foley used a 4 per cent (hypertonic) solution of ferrocyanide and citrate, whereas Weed had used a 1 per cent solution, which is approximately isotonic. Weed [3] and also Nanagas [4] have reported absence of Prussian blue granules within the vessels of the choroid plexus.

If Foley's observations should be confirmed, thus indicating the possibility of a reversal of flow through the choroid plexus, further light might be thrown on the nature of the choroid plexus. With this in mind, we repeated the attempt to demonstrate Prussian blue granules in the vessels of the choroid plexus after intravenous or intraperitoneal injections of hypertonic salt solution, while maintaining intracranial pressure within physiologic limits. The significance of our experiments depends, as do those of Weed, on the assumption that the pathways

1. Weed, L. H.: J. M. Res. **31**:51, 1914; **31**:191, 1923.
2. Foley, F. E. B.: Arch. Surg. **6**:587, 1923.
3. Footnote 1 (second reference).
4. Nanagas, J. C.: Johns Hopkins Hosp. Bull. **32**:381, 1921.

taken by the injected solution and outlined by the precipitated Prussian blue granules truly indicate the physiologic pathways of the cerebrospinal fluid.

Cats under ether, barbital-sodium (sodium diethylbarbituric acid) or amytal anesthesia were used. Barbital-sodium (sodium diethylbarbituric acid), 4 per cent solution, 400 mg. per kilogram by intraperitoneal injection, was given to two animals. Amytal (iso-amyl-ethyl-barbituric acid),[5] 1 per cent solution, 100 mg. per kilogram by intraperitoneal injection, was given to two animals. Ether, by tracheal tube and Woulfe bottle, was given to eight animals.

When completely anesthetized, the animal was placed on its side, lying horizontally, and the subarachnoid space was entered by puncture of the occipito-atlantoid ligament with an 18 gage needle, connected by small-bore rubber tubing to a glass manometer of 1 mm. bore. A buret with a stopcock was connected by an inverted T tube to the cistern needle and to the manometer. The solution was, in all but four instances, the same as that used by Weed and shown by him to be nontoxic: namely, equal parts of potassium ferrocyanide, 1 per cent, and iron ammonium citrate, 1 per cent, freshly prepared. In the four instances mentioned, this solution was diluted with an equal volume of distilled water. The buret, the manometer, the rubber tubing and the needle were filled with the solution to a point approximately 100 mm. above the cistern, so that on puncture no spinal fluid was lost from the subarachnoid space. Pressure readings were recorded every minute, and after a short interval from 20 to 30 cc. of a 30 per cent solution of sodium chloride was injected intravenously or into the peritoneal cavity. Negative pressures within the cranium were readily obtained when the hypertonic solution was given either intraperitoneally or by the intravenous route. The former had the advantage of simplicity and of avoiding a sudden increase in blood volume. The intraperitoneal route was therefore used in most cases. After a transient rise the pressure of the cerebrospinal fluid fell rapidly, and without waiting for it to reach zero more ferrocyanide was slowly run into the cistern from the buret. The total amount of solution run in was usually about 5 cc. (varying from 1.28 to 10.5 cc.) during the course of half an hour. The pressure of the cerebrospinal fluid rose slowly, varying with the speed at which the solution was allowed to run into the cistern from the buret, and an effort was made to keep the pressure below the original readings of the cerebrospinal fluid of the individual animal. This was successful in six instances. In the other six, the pressure rose for a few moments higher than the initial readings. The highest points were 220 mm. of Ringer's solution in one case and 185 mm. in another.

Immediately at the end of the injection, and usually while the heart was still beating, the thorax was opened and from 10 to 20 cc. of a solution of formaldehyde (10 per cent of the commercial solution of formaldehyde) and hydrochloric acid (2 per cent) was injected by syringe into the aorta, avoiding excessive pressure. That the intracranial pressure was not raised to an abnormal height by this injection was shown by leaving the cistern needle in place and watching the pressure of the cerebrospinal fluid in the manometer. In two instances, formaldehyde was poured directly into the ventricles after dividing the corpus callosum without previous intra-aortic injection, in order to avoid the possibility of washing out the ferrocyanide from the vessels by any intravascular addition of fluid.

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5. Eli Lilly & Co., Indianapolis, furnished us with this material for experimental purposes.

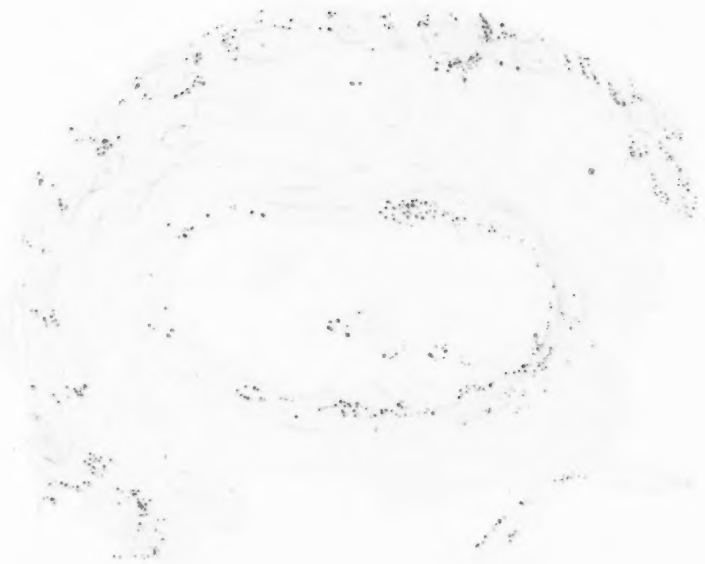

Fig. 1.—Sketch of choroid plexus (oil immersion) showing the distribution of the Prussian blue granules within the lumen and the endothelium of the small blood vessel, and scattered over the epithelial cells. From a section stained with Bismarck brown.

In all cases the brain was removed within two hours and immersed in acid solution of formaldehyde for twenty-four hours.[4] The acid solution was then replaced by neutral solution of formaldehyde and blocks of tissue, including choroid plexus from the lateral and fourth ventricles, were embedded in paraffin. Sections from 3 to 7 microns thick were cut and stained lightly with Bismarck brown or carmalum. Both of these counter stains afforded a good contrast to the blue granules without obscuring them. Examination under oil immersion was found to be necessary for careful identification and localization of the granules, although the large ones could clearly be seen under low power.

As a control for the vessels of the choroid plexus, tissue was taken from other organs: heart muscle, intercostal muscle, lung, liver, spleen and kidney.

Fig. 2.—Photomicrograph with high dry objective of choroid plexus, showing the presence of Prussian blue granules within the lumen of small blood vessels (1), and on the epithelial surface (2). Section stained with carmalum.

It was sectioned, stained and examined in the same way as the sections of the choroid plexus. The purpose of this was to rule out the possibility of the ferrocyanide solution being carried to the capillaries of the choroid plexus through the blood stream.

EXPERIMENTAL OBSERVATIONS

In twelve experimental animals, the several conditions outlined were present.

Histologic examination in every case showed Prussian blue granules in the lumen of the capillaries of the choroid plexuses from both the lateral and the fourth ventricles (figs. 1, 2 and 3). The relative distribution of the granules in the choroid plexus was as follows:

(1) On the epithelial surface—large numbers; (2) between the epithelial cells—frequent in limited areas; (3) in the perivascular regions—occasional; (4) on the inner lining of the vascular endothelium—frequent in certain vessels; (5) within the lumen of veins and capillaries—numerous but often restricted to certain vessels. Occasionally also small arterioles contained granules.

The appearance was not that of a general diffuse infiltration of the tissues due to postmortem alterations described by Weed in cases in which the fixation had been delayed. Such infiltrations were also noted by him when hypertonic solutions of ferrocyanide (5 to 10 per cent) were used. A group of granules found in a capillary of the choroid tuft could often be traced down and shown to form a continuous series with granules in the venule at the base of the tuft, always within its lumen and appearing again and again as the folded vascular

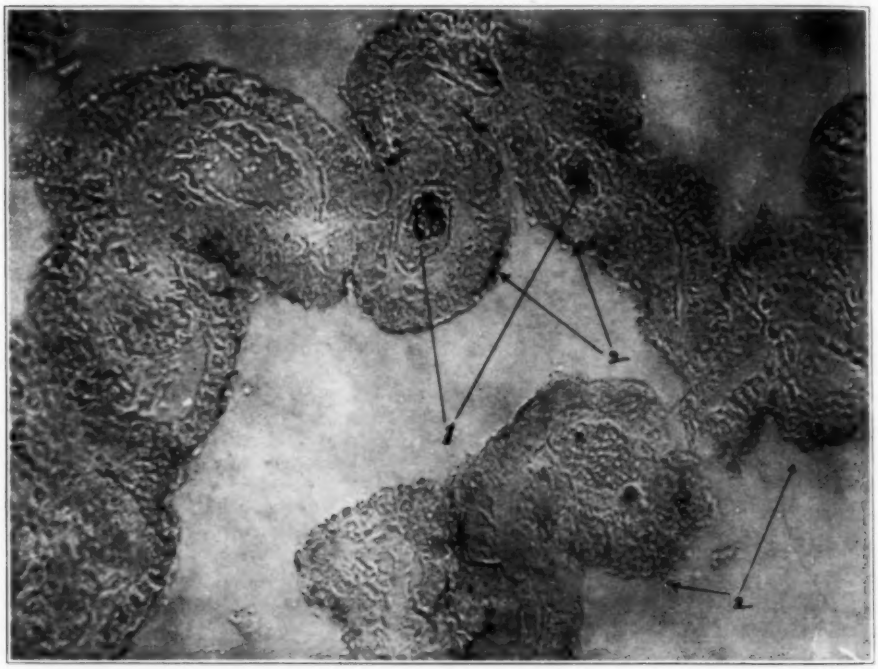

Fig. 3.—Photomicrograph with high dry objective of choroid plexus, showing the presence of Prussian blue granules within the lumen of small blood vessels (1), and on the epithelial surface (2). Section stained with Bismarck brown.

tube was cut in cross or in oblique section. Serial sections showed granules within the same vessels in each section. The finding of granules in arterioles may perhaps be explained by a reversal of flow in the smaller arteries at death. This has been repeatedly observed under the microscope in the pial arteries when the heart stops.[6]

In the sections of tissue taken from other parts of the body, as controls, no Prussian blue granules were found in the liver, spleen, heart muscle or intercostal muscle. Only once in tissue from the lung and three times in tissue from the kidney were occasional granules seen in the finer vessels.

6. Personal observations of Forbes, H. S., and Wolff, H. G.: To be published.

COMMENT

If, as suggested by Weed,[1] the distribution of the granules in the subarachnoid space truly indicates the direction of the flow of cerebrospinal fluid, and the presence of Prussian blue granules in the venous sinuses indicates the normal passage of the cerebrospinal fluid through the membrane of the arachnoid villi, then we feel that the presence of granules within the lumen of capillaries in the choroid plexus indicates with equal justification the passage of cerebrospinal fluid from the ventricles through the choroid plexus into the blood stream under the influence of intravenous hypertonic salt solution. This would indicate that the direction of flow of cerebrospinal fluid through the choroid plexus may be reversed by increasing the osmotic pressure of the blood. Supposing the choroid plexus to be purely a secreting gland, the mechanism of such a reversal of flow would be difficult to understand. If, however, the choroid plexus may be considered a semipermeable membrane through which the direction of fluid exchange is determined largely by the balance between hydrostatic and osmotic forces,[7] a reversal of flow under the foregoing experimental conditions is to be expected.

CONCLUSIONS

Prussian blue granules were found within the vessels of the choroid plexus in each of the twelve animals examined.

This indicates that the direction of flow of cerebrospinal fluid through the choroid plexus may be reversed by increasing the osmotic pressure of the blood. These observations add to the accumulating evidence that the choroid plexus may be regarded as a semipermeable membrane, and the cerebrospinal fluid as a true dialysate.

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7. Fremont-Smith, Frank: The Nature of the Cerebrospinal Fluid Arch. Neurol. & Psychiat. **17**:317 (March) 1927.

CEREBELLAR SYMPTOMATOLOGY EVALUATION ON THE BASIS OF INTRACEREBELLAR AND EXTRACEREBELLAR LESIONS*

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It is generally accepted that the function of the cerebellum is to regulate and coordinate the static and kinetic forces of the organism. Disturbances in this function are at the basis of cerebellar symptomatology. Incoordination or asynergia may, therefore, be said to be the essential symptom of cerebellar disease. Interesting as the various theories of the mechanism of asynergia and its relation to muscle tonus, to deep sensibility and to ataxia may be, the clinician is primarily interested in asynergia as a sign of cerebellar dysfunction. The other clinical manifestations of cerebellar disease—ataxia, dysmetria, adiadokokinesis, hypotonia, tremor, asthenia and cerebellar catalepsy—are merely symptomatic modifications of asynergia.

The conception of the synergic muscular unit, as elaborated by Mills and Weisenburg and later by Lambert and Tilney, has given the clinician insight into the more intimate nature of the anatomic and physiologic factors involved in synergia. A knowledge of these is indispensable to the correct interpretation and evaluation of the symptoms encountered in disease of the cerebellum and the cerebellar pathways.

The object of this communication is to evaluate the cerebellar symptoms in connection with the other clinical features in fifty-one cases of intracranial disease verified by operation or autopsy. We regarded as "verified by operation" only those cases in which the operative results were positive. "Verified by autopsy" refers to macroscopic post mortem observations. We do not have any available evidence as to what these results were in serial sections for purposes of finer localization.

The entire material, clinical as well as pathologic, was collected from the neurologic, surgical, pediatric and otologic services and from the neuropathologic laboratory of Mount Sinai Hospital, from 1920 to 1926 inclusive. It consists of: 19 cases of cerebellar neoplasm verified by operation and 8 verified by necropsy; 2 cerebellar abscesses verified by necropsy; 10 cerebellopontile angle tumors verified by operation and 3 by necropsy; 5 tumors of the brain stem verified by necropsy; 1 tumor

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* From the Neurologic Service of Mt. Sinai Hospital.

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of the fourth ventricle verified by operation, and 3 supratentorial tumors verified by necropsy (1 frontal, 1 temporoparietal and 1 temporo-occipital).

INTRACEREBELLAR LESIONS

The first group discussed consists of twenty-nine cases, (twenty-four tumors of the lateral lobes, two of the vermis and one lateral lobe each, one of the vermis only and two cerebellar abscesses).

Disturbances in Gait.—The most common forms of asynergia were disturbances in gait. These were found in twenty-seven cases; in most of them the gait was described as "cerebellar," "on a broad base," "staggering," "reeling" or "like that of a drunken man." Most of the patients had a tendency to fall while walking. Seventeen fell to the side of the lesion and three to the opposite side; one patient fell to the side of the lesion when he first came under observation but later fell to the opposite side. Two patients had a tendency to fall backward and to the right and left; in one of these the lesion was in the vermis and right lobe, and in the other the lesion was in the vermis and left lobe. Another patient fell back and to the right and left, and his lesion was in the left lateral lobe. Three patients fell in all directions; in one the lesion was in the right lobe, and in the other two in the left lobe. It is also noteworthy that two patients showed nothing peculiar in gait except that they advanced the left shoulder while walking; in one of these the lesion was in the right lobe, and in the other in the left lobe. Another patient with a lesion in the posteromesial part of the right lobe fell only to the right, and still another who had a trunkal type of gait, with falling to the left, showed at necropsy a cyst replacing the entire superior portion of the left cerebellar lobe.

It would seem then that the direction of falling while walking is an important sign in localizing the side of the lesion. This is supported by evidence obtained in experimental and traumatic lesions, limited to the cerebellar lobes. In the usual run of clinical cases, however, the pathologic conditions are not so ideal, and in evaluating this sign it is essential to take into consideration the possible influence of concomitant corticospinal involvement on the gait.

Attitude of the Head and Body.—Disturbances in attitude of the head were noted in sixteen cases. In ten the occiput was directed to the side of the lesion and in six to the opposite side.

In fifteen cases there was falling while in the Romberg position. In nine, the falling was consistently to one side; five of the patients fell to the side of the lesion, and four to the opposite side. The remaining six patients fell to either side, but in two of these the falling was more marked to the side of the lesion. One patient with intense vertigo collapsed as soon as he was placed on his feet. Two others who fell while walking also fell while in the sitting posture, one to the side of the lesion and the other to the opposite side.

Except perhaps for the position of the head, the attitudes while standing and sitting were not sufficiently constant to be as diagnostic of the side of the lesion as was gait. The special attitudes of the trunk described by some observers were noted in only one of our patients, in

whom, while in the Romberg position, the trunk was directed to the right and the occiput to the left. The lesion in this case was in the left lateral lobe.

Ataxia.—The next most common form of asynergia was cerebellar ataxia; it was present in the upper extremity in twenty-five cases. The ataxia was unilateral in twenty-nine cases; it was on the side of the lesion in eighteen, and on the opposite side in two. In five patients it was bilateral, but more marked on the side of the lesion in four.

Ataxia in the lower extremity was present in seventeen cases; it was unilateral in eleven, in nine of which it was on the side of the lesion and in two on the opposite side. It was bilateral in six, and more marked on the side of the lesion in five. In three cases it was present in all extremities and more marked in the extremities on the side of the lesion. In one of the vermis tumors the ataxia was equally marked in both upper extremities and in one lower; in another case with the lesion in the inferior part of the vermis and in the right lobe, the ataxia was in the right upper extremity and in both lower extremities, but more marked in the left lower; in a third case with the lesion involving the vermis, left lobe and left middle peduncle, the ataxia was only in the arms and more marked on the left side.

The ataxia was best manifested by a so-called decomposition of movement in the distal parts of the limbs involved. The preponderance of the ataxia on the side of the lesion in the cases in which it is present, makes it an important sign in localizing the side of the lesion.

Adiadokokinesis.—This symptom was noted in twenty-one cases; it was unilateral in eighteen, and bilateral in three; in fifteen of the unilateral cases it was on the side of the lesion, and in two on the opposite side; in one of the vermis cases it was on the left side. In the case in which the vermis and right lobe were involved, it was present on both sides but more marked on the right side; in another vermis case in which the left lobe and middle peduncle were involved, the adiadokokinesis was present only on the left side. In the bilateral cases it was more marked on the side of the lesion.

Apparently, also, then, the side of the adiadokokinesis is important in determining the side of the lesion.

Hypotonia.—This condition was noted in fifteen cases. It was unilateral and on the side of the lesion in five, and in two it was present in both arms only. In the remaining eight it was present in all extremities; in three of these it was more marked on the side of the lesion.

The hypotonia was in some cases associated with hyperactive reflexes, in others with decreased reflexes and in still others with absent deep reflexes. Likewise, relationship could not be established between the hypotonia and the degree of pyramidal tract involvement in cases in which these two conditions coexisted.

Holmes' Rebound Phenomenon.—This characteristic was present in eight cases. In five it was on the side of the lesion; in two it was bilateral, but more marked on the side of the lesion, while in one case in which the lesion was in the vermis it was present only in the left arm. Only four cases in which this phenomenon was present showed hypotonia; ataxia, however, was present in all eight cases.

Dysmetria.—The condition was noted in seven patients. In four it was on the side of the lesion, and in one on the opposite side. In the remaining two it was present on both sides; in one of these it was more marked on the side of the lesion, and in the other on the opposite side. In every case in which there was dysmetria, ataxia was present in addition; in five it was also associated with hypotonia.

Signs and Symptoms in Fifty-One Cases of Intracerebellar and Extracerebellar Lesions

		Symptoms	Intra-cerebellar	Cerebello-pontile	Brain Stem	Supra-tentorial
		Number of cases	29	13	6	3
Direct	Asynergia	Gait (Disturbance)	27	8	3	2
		Head, attitude (Disturbance)	16	7	2	..
		Station (Disturbance)	15	7	2	1
		Ataxia, upper	25	8	4	3
		Ataxia, lower	17	4	5	2
		Adiadokokinesis	21	8	4	2
		Hypotonia	15	..	3	1
		Holmes' phenomenon	8	5	1	..
		Dysmetria	7	2	..	..
		Tremor	7	3	2	2
		Pendulous knee jerks	4	1	..	..
		Cerebellar speech	5	2	..	2
		Cerebellar catalepsy	2	..	..	..
		Cerebellar fits	3?	..	..	..
	Vestibular	Spontaneous nystagmus	23	13	5	1
		Spontaneous past-pointing	9	3	..	1
Indirect	Cranial nerves	I	..	1	..	..
		II	22	13	5	2
		III, IV, VI	11	9	3	3
		Sensory, motor V	6	11	2	..
		Corneal sensibility disturbed	11	11	4	1
		Supranuclear VII	19	2	2	1
		Peripheral VII	5	10	3	..
		Cochlear VIII	5	13	1	1
		IX, X, XI, XII	5	5	2	..
		Pyramidal, tract signs	24	10	5	3
		Mental sypmptoms	6	5	1	1
Subjective		Headache	25	10	3	2
		Vomiting	21	5	3	2
		Vertigo	13	9	2	2
		Vision, diminished	12	8	1	..
		Diplopia	5	4	2	1
		Gait, attitude, clumsiness	5	9	..	..
		Deafness	3	11	..	..
		Tinnitus	2	7	..	..
		Paresthesias of face	2	6	..	..
		Sphincters	..	2	..	..
		Weakness	3	2	3	..

Tremor.—This symptom was present in seven patients; two had tremor of the head; one of these also had tremor of the arm on the side of the lesion. Four others had tremor in both upper extremities, and one had tremor in the arm and leg on the side of the lesion. The tremor was coarse and of the type usually designated as cerebellar. In five of the cases it was associated with hypotonia.

Cerebellar Catalepsy.—This condition was present in two cases; in one it was on the side of the lesion, and in the other in all extremities. Definite relationship did not exist between cerebellar catalepsy and ataxia, dysmetria or Holmes' rebound phenomenon.

The occurrence of Holmes' rebound phenomenon, dysmetria, and tremor in relatively so small a number of cases, and their almost constant association with ataxia, would seem to confirm the view generally held that they are merely modifications of asynergia, and, as such, only of corroborative value in the diagnosis of cerebellar lesions.

Spontaneous Nystagmus.—This characteristic was noted in twenty-three cases. It was in the horizontal plane in every case, and in nine also in the vertical plane. The slow component was in as many cases to the side of the lesion as to the opposite side.

We could not find any discernible relationship between the degree of increased intracranial pressure and the presence of nystagmus, nor could we find any relationship between it and the degree of asynergia. In our opinion this sign needs further investigation before any diagnostic evaluation can be given as to its precise significance in cerebellar disease, especially in regard to its usefulness as an aid in indicating the side of the lesion.

Spontaneous Past Pointing.—This sign was present in nine cases. Only two of the patients past pointed correctly; in the remaining seven the direction of the spontaneous past pointing was so inconstant as to be without localizing value.

Caloric Examination.—The results are recorded in twenty-two cases. In two the examination was unsatisfactory and inconclusive. The results of the tests were in full accord with the neurologic results in six cases. In two cases the caloric tests wrongly pointed to a lesion in the lobe opposite to that indicated by the neurologic observations. In four the vestibular tests were reported as normal, but the neurologic observations pointed definitely to a lesion in a cerebellar lobe, in which it was found at operation. In two cases the neurologic observations pointed to a lesion in the cerebellopontile angle and the cochlear and vestibular tests confirmed this localization; at operation the lesion was found in each case in a cerebellar lobe and not in the angle. Two patients showed clinical evidences of a posterior fossa lesion; the vestibular tests confirmed the diagnosis of a posterior fossa lesion but did not shed further light as to the localization. In one case the signs on admission pointed to a lesion in the left cerebellar lobe; three successive vestibular tests, done within one month, reported "nothing localizable;" a fourth examination, two months later, pointed to a "nonlocalizable posterior fossa lesion;" at operation the lesion was found in the left lobe. In another patient the neurologic tests pointed to a tumor in the left cerebellar lobe; the caloric tests confirmed this localization, but when the tests were repeated two days later, they were reported as indicating a lesion in both cerebellar lobes; at operation the tumor was found in the left lateral lobe. Finally, in one case the neurologic observations pointed to a lesion in the posterior fossa, "side not localizable;" the otologist localized the lesion correctly. (All these tests were done by the otologist to the hospital.)

The vestibular tests have proved important in differentiating labyrinthine from intracranial disease. In some patients with tumors of one or the other cerebellar hemispheres the reactions are sufficiently charac-

teristic to be regarded as confirmatory evidence in localizing the disease; in other patients with intracerebellar disease the results are often ambiguous and do not afford any aid in establishing a localization, and, taken by themselves, may even lead to erroneous conclusions.

Involvement of the cranial nerves, other than the vestibular, does not belong to the direct symptomatology of intracerebellar disease. In many cases, however, cranial nerve involvement develops during some stage of the disease. Many of our patients showed involvement of the third, fourth and sixth nerves. Little reliance, however, can be placed on their localizing value.

Papilledema.—This condition was present in twenty-two cases. Eight of these showed more marked papilledema on the side of the lesion, and three on the opposite side. In eight the elevation of the disks was equal on the two sides; in two patients with lesions in the vermis and in one lateral lobe, the edema was more marked on the side of the lobe involved in one, and in the other it was more marked on the opposite side; in another patient with a lesion in the vermis only, the papilledema was equal on the two sides. Five patients showed various degrees of postneuritic atrophy prior to operation, and three after operation. In four cases the papilledema was associated with hemorrhages in the disk. A fairly constant relationship existed between the disk changes and the intracranial pressure.

Trigeminus.—This nerve was involved in six cases. In three only the sensory divisions were affected, in two on the side of the lesion and in one on the opposite side. In one, both the motor and sensory divisions were affected on the side of the lesion, and in one on the opposite side. In one with the lesion in the right lobe the sensory involvement was on the left side, and the motor on the right. In four of the cases with trigeminus involvement the intracranial pressure was noted as markedly increased, in one it was moderately increased and in one there was no evidence of increased intracranial pressure.

Diminution or absence of corneal sensibility was noted in eleven cases; in seven it was bilateral and in four unilateral. It was bilateral in two of the vermis cases, and unilateral in the third vermis case. When the involvement was unilateral, it was on the side of the lesion in three, and on the opposite side in one. In nine of the eleven cases there was evidence of increased intracranial pressure. In only two cases was the disturbance in corneal sensibility associated with involvement of the sensory fifth nerve.

Facial.—In twenty-four cases the innervation of the facial muscles was faulty; in five of these it was peripheral and in nineteen supranuclear. The peripheral facial lesions were slight and often variable and inconstant; it was on the side of the lesion in three, and on the opposite side in two. Supranuclear facial involvement was associated with pyramidal tract involvement in seventeen of the nineteen cases; in the other two it was inconstant and as variable as in the peripheral cases.

Cochlear.—Nerve deafness was present in five cases; it was unilateral in four and bilateral in one; in the latter the lesion was in the right cerebellar lobe; in three of the former the cochlear involvement was on the side of the lesion; in the fourth the lesion was in the vermis. In three cases the nerve deafness was total; in two of these the vestibular reactions with the clinical picture was such as to lead to exploration of the cerebellopontile angle, and in each case a

tumor was found in the cerebellar lobe. Four of the five patients with cochlear involvement showed definite increase in intracranial pressure.

Other Nerves.—The remaining cranial nerves were involved as follows: Three patients had pharyngeal anesthesia and difficulty in swallowing; two others showed weakness of the soft palate on the side of the lesion. Of the three cases of difficult swallowing one occurred in the patient with the lesion in the vermis and right cerebellar lobe. All these cases showed evidences of marked intracranial pressure.

Speech Disturbances.—These defects, recorded as "cerebellar," "staccato," "scanning" and "explosive," were noted in five cases.

Pyramidal Tracts.—Involvement of the pyramidal tracts was found in twenty-four cases. In twenty-one the involvement was unilateral, and in three it was bilateral; in one of these the lesion was in the vermis and right lobe; in the twenty-one unilateral cases the lesions were on the side of the cerebellar involvement in sixteen, and on the opposite side in five.

Although all except one of the cases with pyramidal tract signs showed evidences of increased intracranial pressure, there was not any relationship between the severity of the latter and the intensity of the former.

One case showed total areflexia of the deep and diminution of the superficial reflexes, and four showed diminution of all reflexes; in the former the intracranial pressure was unusually increased, and in the latter markedly increased. There were two cases with increased intracranial pressure without changes in the reflexes. The changes in the deep reflexes were not always associated with corresponding changes in the superficial reflexes; the same was true as regards the presence or absence of pathologic reflexes.

The daily variability in the reflexes was a striking feature. This was probably due to the periodic variations in the intracranial pressure.

A pendulous knee jerk was noted in four cases; in three it was unilateral and on the side of the lesion, and in one it was bilateral; in the latter the lesion was in the vermis, left lobe and left middle peduncle. The pendulous knee jerks all occurred in patients with marked hypotonia.

In evaluating these facts we believe that we are justified in stating that the great variability in the reflexes minimizes their value as a localizing sign. We feel that by themselves the reflexes are not to be taken into consideration in the localization of intracerebellar lesions. Our cases show that in at least two instances inclusion of the reflex changes in the clinical picture, with the view of their being an aid in the localization, led to a wrong interpretation. We feel therefore that, given a case with signs and symptoms pointing to a lesion in the posterior fossa with all evidences pointing to intracerebellar involvement, the localization of the side of the lesion will depend on the site of the cerebellar signs and not on the pyramidal tract signs. The presence of pyramidal tract signs and other changes of the reflexes will best be considered as indirect signs and probably indicative of increased intracranial pressure or extension of the process into neighboring structures.

Mental Symptoms.—These symptoms were noted in six cases and were similar to those ordinarily encountered in intracranial tumors.

Seizures.—One patient had, before admission, two attacks of rigidity which involved the entire body. We are unable to state whether or not these were "cerebellar fits;" another patient had several general convulsions with loss of consciousness before and after operation, and still another had "tonic" and "clonic" convulsions.

Age, Sex and Duration of Illness.—The group was composed of twenty male and nine female patients. The ages varied between 4 and 55 years. Thirteen patients were in the first decade of life—of these, four were 4 years of age; seven were in the second; two in third; four in the fourth, and three in the fifth decade of life.

The duration of the illness before admission was said to be one month in one case, two months in four cases, three months in five cases, four months in three cases, five months in two cases, eight months in one case, ten months in one case, twelve months in one case, two years in one case and four years in one case; in one case the onset was sudden, and in another the date of onset was unknown.

Subjective Symptoms.—Headache was present in twenty-five cases; it was the first symptom in eighteen; it was generalized in nineteen, frontal in three, fronto-occipital in two and temporoparietal in one. Vomiting occurred in twenty-one cases, and was associated with headache as the first symptom in twelve patients. Vertigo was noted in thirteen cases and was the first symptom associated with headache in four; it was the only first symptom in twelve patients. Stewart and Holmes state that the vertigo in intracerebellar tumors is usually described as subjective vertigo from the side of the lesion to the healthy side. The transient nature of the vertigo in our cases and the mental confusion accompanying it, associated with the facts that 61 per cent of our patients were in the first decade of life, and that the general intelligence of the group was such that we did not consider them sufficiently reliable to permit us to determine in which direction the subjective vertigo was, have resulted in our not having any data on this point.

General weakness was associated with headache as a first symptom in three patients, but all of them complained of persistent vomiting. Headache and diplopia were noted as first symptoms in five cases. Headache with cyanosis was the first symptom in one case. Paresthesia of the face was the first symptom in two cases. Disturbance in gait and attitude, and clumsiness, associated with headache, were the first symptoms in five cases. Diminished vision was complained of by twelve patients. Diminished and dim vision, associated with headache, were the first symptoms in seven cases. Speech and gait disturbances were the first symptoms in one case. Three cases began acutely with fever, headache and vomiting, so that in two the diagnosis of encephalitis was made prior to admission, and in one meningitis was diagnosed. Two patients complained of tinnitus and deafness, and one of deafness without tinnitus, before admission. Two others complained of somnolence only.

CEREBELLOPONTILE ANGLE CASES

Disturbances in Gait.—These disturbances were present in eight of the thirteen patients in this group. Three walked on a broad base and fell toward the side of the lesion; one fell backward and toward the side of the lesion; one fell backward and to the right and left, the lesion being on the left side; one patient with a bilateral lesion, more massive on the left side, fell to both sides but more to the left; another patient with the lesion on the right, fell to the

right and left, but more to the right, and one fell to the side opposite to the lesion. In one patient with the lesion on the right side, gait and station were normal, but she walked advancing the right shoulder.

Attitude of the Head and Body.—Disturbances were noted in seven cases; in these the occiput was turned to the side of the lesion in two and to the side opposite the lesion in four; and in one case, a bilateral lesion, it was turned to the side of the more massive lesion.

In seven cases there was swaying and falling in the Romberg position; four of the patients swayed and fell to the side of the lesion; one swayed in all directions; one swayed to the right and left and forward, and one swayed to the right and left and backward.

Twelve of the thirteen cases, then, showed disturbances in the position of the head, in gait or while in the Romberg position. Relationship could not be established between the direction of falling, the attitude of the head and the side of the lesion. Apparently, disturbances in gait in cerebellopontile angle lesions are not nearly as valuable in indicating the side of the lesion in cerebellopontile angle as in intracerebellar lesions.

Ataxia.—Eight patients showed ataxia. Four of them had ataxia only in the upper extremities, and four others in both the upper and lower. The ataxia in the upper extremities was on the side of the lesion in six; in the remaining two it was bilateral, but more marked on the side of the lesion. In the four cases with ataxia in the upper and lower extremities, the ataxia in the lower was on the same side as that of the upper in three, and on the opposite side in one. The patient with the bilateral tumors did not show any ataxia.

The ataxia in the cerebellopontile angle cases was, relatively speaking, less frequent than in the intracerebellar cases, but when present was more marked on the side of the lesion.

Adiadokokinesis.—This condition was found in eight cases; it was unilateral in seven, and in six of these was on the side of the lesion; in the patient with the bilateral tumors it was on the side of the more massive lesion; in one case it was bilateral, but more marked on the side of the lesion. There does not seem to have been any relationship between the degree of ataxia and adiadokokinesis.

This form of asynergia was, relatively speaking, as frequent as in the intracerebellar cases. Its invariable presence on the side of the lesion gives it importance as a localizing sign.

Hypotonia.—This characteristic was not observed in any of the cerebellopontile angle cases; in one case there was a pendulous knee jerk on the side of the lesion.

Holmes' Rebound Phenomenon.—This sign was present in five patients, on the side of the lesion in all; in two it was found in the arm only, and in three in the arm and leg.

Dysmetria.—This symptom occurred in two cases, also on the side of the lesion.

Three cases did not show any ataxia, hypotonia, adiadokokinesis or dysmetria.

Tremor.—One patient had a slight tremor of the head, another, a tremor of the head and both hands and a third, an ataxic tremor of the right hand; in this case the lesion was on the right side.

The degree of asynergia in the cerebellopontile angle cases was never as marked as in the cases of intracerebellar disease.

Spontaneous Nystagmus.—This characteristic was present in every case. In nine the slow component was to the side of the lesion, and in six of these there was also some nystagmus in the vertical plane; in four of the latter there was also a rotatory element to the nystagmus. Two cases showed the slow component to the side opposite the lesion; in two others the nystagmus was of the same amplitude in both lateral planes; in one of these there was also nystagmus in the vertical plane. In the patient with the bilateral lesions the slow component was to the side of the more massive lesion.

Spontaneous Past Pointing.—This sign was noted in three cases. In only one patient was the past pointing correct; in the other two the direction of the past pointing was so inconstant that it was impossible to evaluate this sign.

Caloric Tests.—These tests were done in twelve cases; in ten the results of the tests confirmed the clinical diagnosis of cerebellopontile angle neoplasm. It may, perhaps, be of interest that in only one of these cases was the caloric response typical of a cerebellopontile angle tumor, and in another patient it was atypical before operation, but became typical one month later. In one case the clinical diagnosis was tumor in the left angle, where it was found at operation, and the otologist resported that the tests indicated a lesion "other than an angle tumor." In another case the caloric tests indicated a lesion in the right cerebellar lobe, and the neurologic observations were those of a tumor in the right angle, where it was found at operation.

In evaluating the results of caloric tests, we do not hesitate to state that they are considerably more valuable and reliable in localizing cerebellopontile angle than intracerebellar lesions.

Cranial Nerves.—One patient with a tumor in the right angle had loss of the sense of smell on that side and impairment on the other. Four cases showed unequal pupils. The anisocoria did not bear any relationship to the disk changes. The extra-ocular muscles were involved as follows: One patient with a tumor in the right angle was unable to converge the right eye; one patient with a bilateral lesion had insufficiency of both internal recti, and weak upward gaze on the side of the more massive lesion; one had involvement of the left superior rectus muscle, the lesion being on the left side. Nine patients showed involvements of the sixth cranial nerve; it was unilateral in seven and bilateral in two; in all unilateral cases, the involvement of the sixth nerve was on the side of the lesion. Skew deviation was observed in one case, and, as in one of the intracerebellar cases, before operation. Definite relationship did not exist between the degree of intracranial pressure and the involvement of any of the extra-ocular muscles, except that in the case with skew deviation the pressure was enormously increased.

Changes in the fundi were observed in every case. They varied in the different cases from slight blurring of the disk with fulness of the veins to 5 diopters elevation. The papilledema was equal on the two sides in seven; it was greater on one side than on the other in six; in four of these it was greater on the side of the lesion, and in two on the opposite side.

Three cases showed advanced bilateral postneuritic optic atrophy in addition to papilledema; the atrophy was far advanced on admission. Only one case showed minute hemorrhages with the papilledema. The disk changes were in harmony with the degree of increased intracranial pressure.

The trigeminus was affected in eleven cases. In five of these there was involvement of both motor and sensory divisions; in five others only the sensory division, and in one only the motor division. In ten cases the involvement was on the side of the lesion; in one, with motor involvement only, it was on the side opposite the lesion. In the patient with bilateral lesions the trigeminus involvement was on the side of the more massive lesion; here the sensory disturbance was only in the lower two branches of the nerve.

Eleven cases showed diminution or absence of corneal sensation; it was unilateral in eight, in seven of which it was on the side of the lesion; in three it was bilateral, the lesion being on the left side in two, and on the right side in one. In the patient with the bilateral lesions there was corneal hypesthesia on the side of the more massive lesion.

Facial involvement was noted in twelve cases; in eight it was peripheral in type, and on the side of the lesion; in the patient with the bilateral lesions it was also peripheral and on the side of the more massive lesion; in another peripheral case it was on the side opposite to the lesion, but in this patient there had been an old suppurative process in the middle ear on that side; in the remaining two it was supranuclear and on the same side as the pyramidal tract signs, which were on the side opposite to the lesion; in one it was questionable whether the facial involvement was supranuclear or peripheral, and, as it was slight and on the opposite side to the lesion, no evaluation was given to it. In eight cases the peripheral facial palsies were definite; in two others the involvement was slight and it was difficult to decide whether it was central or peripheral, but under observation and with the aid of electrical tests the peripheral nature became apparent.

Nerve deafness was present in every case. It was unilateral and on the side of the lesion in twelve, and bilateral in one. It was total in eleven and partial in two. In the patient with bilateral tumors it was total on both sides.

The remaining cranial nerves were involved as follows: The soft palate was paretic on the left side—the side of the lesion—in two cases; in one the uvula deviated to the left (side of the lesion); all three cases, however, had pyramidal tract signs on the side of the involvement of the palate and uvula. One patient had difficulty in swallowing one year before operation; another developed difficulty in swallowing, one day after operation; this was associated with a complete palsy of the left side of the larynx and an abductor paralysis of the right side. Two patients had atrophy and fibrillary twitchings of the tongue on the side of the lesion. The patients with pyramidal tract involvement had deviation of the tongue to the side on which the pyramidal tract signs were present. The patient with involvement of the motor and sensory fifth, seventh and twelfth nerves had drooling of saliva.

Speech.—Disturbances in speech were noted in three cases. These were described as "cerebellar" in two, and "dysarthric" in one.

Pyramidal Tracts.—Signs of pyramidal tract involvement were noted in ten cases; they were marked in eight and slight in two; they were unilateral in eight, and bilateral in two. In those with bilateral signs they were more marked on the side of the tumor; in the cases with unilateral signs, six were on the side of the lesion and two on the opposite side.

Relatively speaking, pyramidal tract involvement was more frequent in the cerebellopontile angle than in the intracerebellar cases. The periodic variability in the reflexes noted in the intracerebellar group was not observed in the cerebellopontile angle cases.

Mental Changes.—These signs were noted in five cases. One patient had periods of confusion and was irrational on several occasions. Another apparently had several psychotic episodes during the fifteen years of illness prior to admission, and became depressed after operation; another developed a manic state fifteen days after operation, and still another was dull and depressed for months before admission.

Incontinence.—Two patients had urinary incontinence; one for six months, and the other for five weeks before admission.

Age, Sex and Duration of Illness.—There were six male and seven female patients; the ages (in decades) on admission were: second decade, one; fourth decade, four; fifth decade, six, and sixth decade, two.

The duration of illness before admission was: two months in three cases; three months in one case; six months in one case; nine months in one case; two years in two cases; five years in one case; fourteen years in one case; fifteen years in two cases, and twenty years in one case.

Subjective Symptoms.—Headache was present in ten cases; it was frontal in four and generalized in six. It was the first symptom in seven cases.

Vomiting occurred in five cases and was associated with headache as the first symptom in two.

Vertigo was present in nine cases and was associated with headache as the first symptom in two. In this group, as in the intracerebellar cases, it was impossible to determine the direction of the vertigo. One patient with vertigo as the first symptom never complained of headache.

Diminution of vision occurred in eight patients before admission; in two of these it was the first symptom in association with headache. Four patients complained of diplopia. Paresthesia of the face were present in six cases.

Tinnitus was a symptom in seven cases; in one it preceded the deafness by twelve years and in the other six, it appeared simultaneously with the deafness.

Deafness was complained of by eleven patients, in one of whom it appeared three and one-half years before the tinnitus.

General weakness was an early symptom in two patients, and weakness in one or more extremities in another. Weakness was associated with headache as a first symptom in two cases and with vertigo as a first symptom in one case.

Disturbances in gait and attitude, and clumsiness, were subjective symptoms in nine patients, before admission.

As a rule, the general symptoms of tumor of the brain appear earlier and are more pronounced in intracerebellar than in cerebellopontile angle cases. In the former the cranial nerves are less impaired, and are involved later than in the latter. Deafness appears early and is almost invariably present in cerebellopontile angle tumors. Tinnitus and deafness, associated with involvement of the trigeminus and of the facial nerve (peripheral) on the same side, are early and almost constant symptoms in cerebellopontile angle neoplasms. The early onset and greater intensity of the cerebellar symptoms, especially of disturbances

in gait and ataxia, is in favor of an intracerebellar lesion. The presence of cerebellar signs on one side and of pyramidal tract signs on the opposite side is more in favor of an extracerebellar lesion compressing the pons. The symptoms in cerebellopontile angle cases depend a great deal on the size and direction of the growth. In cases in which the tumor begins in the cerebellum the cerebellar symptoms are always more marked than in the case in which the tumor begins in the angle itself.

BRAIN STEM LESIONS WITH CEREBELLAR SYMPTOMS

This group consists of six cases of tumors of the pons and medulla. Five of these were proved by necropsy, and one, a tumor of the fourth ventricle, by operation. For the purposes of this contribution we have designated this group as the "brain stem group with cerebellar symptoms."

Disturbances in Gait.—These signs were present in five patients; they were "cerebellar" in three, and spastic in two. One patient (fourth ventricle tumor) walked holding the head (occiput) to the left and the trunk to the right, and staggered to the left. One had an unsteady gait, walking with the trunk bent forward at the hips with a slight tilt to the left, the legs being set widely apart. One patient staggered to the left. One had a hemiplegic gait and favored the leg on the side of the pyramidal tract signs, and one had a spastic gait in the right leg with a tendency to fall to the left.

Disturbances in Attitude.—These conditions were noted in four cases. Two patients were unable to stand in the Romberg position and fell in all directions. One patient held the head (occiput) to the right, and one (fourth ventricle tumor) to the left, while walking.

Ataxia.—This defect was present in five cases; it involved all four extremities in three; both lower extremities in one patient, and the upper and lower extremity on the same side in another. The patient with the tumor in the fourth ventricle did not have ataxia.

Adiadokokinesis.—This symptom was present in four cases; it was unilateral in three, and bilateral in one.

Hypotonia.—This condition was present in three cases; it was unilateral in two and bilateral in one. The patient with the fourth ventricle tumor had hypotonia in both upper extremities only.

Holmes' Rebound Phenomenon.—This sign was observed in only one case, and it was more marked on one side of the body than on the other.

Tremor.—Two patients had a coarse tremor of the hand while at rest which became more marked on intention. One patient showed an ataxic tremor in the left upper extremity.

The patient with the fourth ventricle tumor showed the following cerebellar signs: spontaneous nystagmus, cerebellar gait, cerebellar attitude of the head and trunk, hypotonia of both upper extremities and cerebellar speech.

The cerebellar symptoms in all patients in this group were not as definite as in the preceding groups. This was especially true as regards

the ataxia and adiadokokinesis. The complicating pyramidal tract signs in most of the cases made it difficult to estimate whether the clumsiness was due to asynergia or to pyramidal tract involvement.

Cranial Nerve Involvement.—In this group there was anisocoria in four cases. In one patient with an extensive pontile lesion both pupils were fixed to light and reacted well in accommodation; he showed neither serologic nor pathologic evidences of syphilis. One patient had paresis of the superior rectus and an abducens weakness on one side; another had weakness of the left internal rectus and paralysis of both abducentes.

Optic nerve changes were noted in five cases. One patient showed on admission blurred disks, which progressed to a swelling of 3 diopters within a month, that is, six weeks after the onset of the illness; in this case the cerebrospinal fluid was under normal pressure, and there was not any evidence of increased intracranial pressure at necropsy. One patient, whose illness lasted two years, did not show any changes in the disks until some weeks before death when he developed a mild optic neuritis, with some pallor of the disks. One patient was admitted with normal nerve heads and within six weeks developed bilateral papilledema of 2.5 diopters; at necropsy the brain showed evidences of markedly increased intracranial pressure. One patient showed on admission (one month after the onset of the illness) bilateral papilledema of 4 diopters; the brain at necropsy also showed evidences of markedly increased intracranial pressure. One patient with evidences of markedly increased intracranial pressure had normal disks throughout the illness, which lasted approximately two and one-half months. The patient with the fourth ventricle tumor was admitted with bilateral papilledema of from 3 to 4 diopters and beginning optic atrophy, the duration of the illness being six months at the time of admission.

The fifth motor cranial nerve was involved in two cases; it was unilateral in both. The sensory division of the fifth was intact in all six cases.

Disturbances in corneal sensibility were noted in four cases; it was bilateral in three, and unilateral in one.

Disturbances in facial innervation were noted in five cases; peripheral in three, and supranuclear in two in association with pyramidal tract signs; in one case, the supranuclear involvement was on the side of the pyramidal tract signs, and in the other case, with evidence of bilateral pyramidal tract involvement, it was on the side of the more marked pyramidal tract signs.

Cochlear involvement (unilateral) was noted in one case.

Spontaneous Nystagmus.—This symptom was noted in five cases; in four it was present in both horizontal planes, and in two of these it was also vertical and oblique; in the case with the fourth ventricle tumor, spontaneous nystagmus was noted only on looking to the right.

Spontaneous Past Pointing.—None of the patients in this group past-pointed spontaneously.

Caloric Tests.—These tests were performed in four cases. They were unsatisfactory in the patient with the fourth ventricle tumor; in another case definite conclusions could not be drawn; in the remaining two, the tests corroborated the clinical diagnosis of a stem lesion.

Difficulty in Swallowing and Dysarthria.—These signs were noted in one case. In another, there was involvement of the ninth and tenth on one side, and of the eleventh and twelfth cranial nerves (peripheral) on both sides.

Pyramidal Tract Signs.—These characteristics were present in five cases (in all patients except the one with the fourth ventricle tumor); they were bilateral in three, and unilateral in two. None of the cases showed a clearcut paralysis alternans.

Mental Symptoms.—One patient had euphoria and emotional instability.

Age, Sex and Duration of Disease.—This group consisted of three male and three female patients. Four patients were in the first decade, one was in the third and one was in the fourth. The youngest patient was 3 and the oldest 39 years of age.

Subjective Symptoms.—In one case, the history of the mode of onset could not be obtained. Of the remaining five patients, three had generalized headache, and two others had vertigo; three complained of weakness of the extremities; two had persistent vomiting; two complained of double vision, and another of poor vision. A noteworthy feature in this group was the relatively early appearance of the general symptoms of brain tumor.

A review of the histories and clinical signs of the cases in this group, in the light of the postmortem results, does not permit definite conclusions. Some of the outstanding features may be said to be: the predominance and earlier appearance of the pyramidal tract and cranial nerve signs over the cerebellar signs; the asynergia is, as a rule, less definite in quality and situation; the cranial nerve symptoms are often bilateral and more general in the brain stem cases than in the intracerebellar and cerebellopontile angle cases. Our pathologic and clinical data in this small group of cases do not give any clues as to what one is to expect clinically from involvement of any of the cerebellar peduncles. The nature of the cerebellar symptoms, in the light of our personal experience and from what we could gather from the literature, cannot be utilized as a diagnostic factor in an attempt to postulate, with any reasonable degree of certainty, the localization of lesions in the peduncles. The diagnosis must be made, not on the nature and distribution of the cerebellar symptoms, but on their association with other symptoms due to involvement of the pons and medulla, and on the history and chronologic appearance of each symptom.

SUPRATENTORIAL CASES WITH CEREBELLAR SYMPTOMS

To illustrate the difficulties encountered clinically in evaluating cerebellar symptoms in cases of intracranial neoplasms we cite, in more or less detail, three cases:

REPORT OF CASES

CASE 1.—A man, aged 40, was admitted to the hospital with a history of attacks of vertigo, loss of memory and emotional instability of three years' duration. On examination he showed: sluggish pupils; left hemiparesis; bilateral, low grade optic atrophy; a cerebellar tremor, ataxia and adiadokokinesis in both upper extremities, more marked in the left; hypotonia on the left side, and a monotonous staccato speech. The frontal region was explored and a neoplasm was not found.

Necropsy showed an endothelioma, 2 inches (5 cm.) in diameter, involving the dura in the midline between the inferior surface of both frontal lobes and compressing the olfactory tracts; the tumor infiltrated the right frontal lobe and impressed itself into the left frontal lobe; it had penetrated for about 1½ inches (3.8 cm.) into each frontal lobe. The corpus callosum was not involved; the ventricles were small and not disturbed. Another tumor, 1 cm. in diameter, was found on the dura overlying the anterior part of the petrous portion of the left temporal bone; it had also penetrated through the dura.

CASE 2.—A girl, aged 16, was admitted to the hospital complaining of vertigo and nausea of five months' duration, after which she developed diplopia, slowness of speech and a staggering gait with paresthesias of the right side of the body. During this period she had one convulsion (generalized) after which she was semicomatose for three days. Two weeks before admission she noticed difficulty in hearing, and one week later she had an olfactory hallucination. She had lost 10 pounds (4.5 Kg.) during the month prior to admission.

Examination revealed: normal fundi; complete right hemiparesis with bilateral ankle clonus; right pupil larger than left; some vertical nystagmus; right homonymous hemianopia; skew deviation of the eyes; bilateral corneal hypesthesia; definite bilateral ataxia and adiadokokinesis, more marked on the right; falling to the right while walking, and a positive Romberg sign. The spinal fluid came out under increased pressure. Ventriculography showed obliteration of the left lateral ventricle and dislocation of the right lateral and third ventricles.

Operation revealed a large infiltrating glioma in the left cerebral hemisphere. The tumor presented itself on the surface in two places: (1) between the frontal and parietal lobes posteriorly to Broca's area, and (2) beneath the surface, pushing up the brain in the occipital lobe at the supramarginal terraces; it was not removable.

CASE 3.—A man, aged 41, was admitted to the hospital with an "acute" history of three days' duration, of vertigo, excruciating headache, pain in the occipital region, rigidity of the neck, tremor of the hands and persistent projectile vomiting. On admission, the pulse rate was 60 and the temperature 100 F., and he was in semistupor.

Examination revealed: left pupil larger than the right; normal fundi; slight bilateral external rectus weakness; slight right supranuclear facial weakness; hyperreflexia on the right side; marked rigidity of the neck, and a bilateral Kernig's sign. Lumbar puncture yielded a bloody fluid under moderate pressure.

The patient's condition remained stationary for two days, after which he was suddenly seized with a "tonic" fit, associated with loss of consciousness; during the fit the entire body assumed a typical decerebrate rigidity; during the next two days he had three similar seizures. Following the last seizure the gait became ataxic and he developed marked asynergia in all four extremities, horizontal and vertical nystagmus, and a coarse head tremor. The vestibular tests were reported as indicating an intracerebellar lesion. He remained in this condition for six weeks after which he began gradually to improve; after two more weeks he had practically recovered, except for some slight ataxia in the upper and lower extremities. He was quite comfortable for the next two months, and then headache and vertigo recurred and the ataxic gait became more marked.

Examination at this time revealed: left pupil larger than the right; early bilateral papilledema; left homonymous hemianopia; left astereognosis; disturbances in postural sensibility in the limbs on the left side, and left hemiparesis with an equivocal plantar response on both sides. The cerebellar signs

were not as definite as at the onset of the illness. The spinal fluid was clear, under moderate pressure, and contained 24 lymphocytes. The general condition became progressively worse, and he died about five months after the onset of the illness.

At necropsy the brain showed evidences of increased intracranial pressure. A tumor (spongioblastoma) 7 by 4 cm. was found on the inferior surface of the right temporo-occipital lobes; it lay posteriorly to the thalamus and mesially to the optic radiation; it had broken through to the surface on the mesial side of the right occipital lobe, dislodging the mesencephalon to the opposite side, and it compressed the brain stem in the region of the quadrigeminal plate; the aqueduct of Sylvius was reduced to an oblique slit. The tumor also invaded the posterior horn of the left lateral ventricle, part of which was collapsed.

The occurrence of cerebellar symptoms in supratentorial lesions other than those illustrated by the cases described (frontal, suprasellar, temporal, etc.) is too well known to necessitate comment. Neurologic literature teems with descriptions of various criteria for the differentiation of so-called frontal lobe ataxia, temporal lobe ataxia, rubral ataxia, hyptokinesis, etc., from the ataxia due to disease of the cerebellum itself. We have not been successful in making such differentiation at the bedside from the nature of the cerebellar symptoms, and are, therefore, unable to confirm or deny the tenability of the various differential criteria. As a matter of fact, the three cases cited in detail and selected at random from the available clinical material illustrate the exception to the well established rule that cerebellar symptoms in supratentorial lesions appear late, whereas those in posterior fossa lesions are usually present from the onset of the illness. In each of the three cases the clinical picture began with cerebellar symptoms and signs. A correct evaluation of cerebellar symptoms produced by lesions in localizations other than those of the cerebellum itself, from the history and clinical observations, may frequently be almost impossible. In such cases encephalography is of the greatest aid in reaching a diagnosis.

FORCED CONJUGATE UPWARD MOVEMENT OF THE EYES FOLLOWING EPIDEMIC ENCEPHALITIS

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Under the title of "Forced Conjugate Upward Movements of the Eyes," Hohman [1] has described, for the first time in this country, an unusual disturbance following epidemic encephalitis and apparently associated with the postencephalitic parkinsonian syndrome. The symptom consists essentially of a forced upturning of the eyeballs, coming on in intermittent attacks and lasting a varying time. There are no discoverable constant subjective symptoms associated with the spasmodic movement, and it obviously cannot be classified as hysterical. Hohman reviewed what little is known of the subject, and drew attention to articles by Fischer,[2] Meyer,[3] Ewald,[4] and also to one on a closely related aspect of the subject by himself,[5] which showed that the condition had been observed in Germany for almost a year and that, exclusive of the American cases, twelve instances of the condition had been published.

Various other papers have also appeared in France and in Germany during the past two years describing cases of deviation of the eyes following encephalitis, some of which are evidently of the same type as that described by Hohman, while others are closely allied. As an increasing number of cases is reported, it is becoming evident that, although the upward deviation of the eyes appears to be the commonest spasmodic movement, others may also occur. Kulkow [6] has published the report of a case in which the spasm was toward the left, occurred once in three or four days, was of from two to three hours' duration, and was relieved by sleep. Bériel [7] reports an instance of deviation upward and to one side, which was also relieved by lying down. In 1924, Lemos [8] published the report of a case, under the general title of "Intermittent Claudication," in which various cramps of the muscles of mastication and of the

1. Hohman, L. B.: Forced Conjugate Upward Movements of Eyes in Postencephalitic Parkinson's Syndrome, J. A. M. A. **84**:1489 (May 16) 1925.
2. Fischer, B.: Deutsche Ztschr. f. Nervenh. **164**:1924, 1924.
3. Meyer, A.: Arch. f. Psychiat. u. Nervenh. **70**:500, 1924.
4. Ewald, G.: Monatschr. f. Psychiat. u. Neurol. **57**:222, 1924.
5. Hohman, L. B.: Arb. a. d. neurol. Inst. a. d. Wien. Univ. **27**:1, 1925.
6. Kulkow, A. A.: Ztschr. f. d. ges. Neurol u. Psychiat. **102**:636, 1926.
7. Bériel, L.: Lyon méd. **136**:45, 1925.
8. Lemos, M.: Rev. neurol. **40**:425, 1924.

arms occurred in association with conjugate deviation of the head and eyes, all of which he regarded as due to a crisis of extrapyramidal hypertonia, the lesion being presumably in the striatum. Popowa [9] alludes to both clonic and tonic spasm, also to rhythmic tonic spasm drawing the eyes upward, and speculates regarding the seat of the disturbance. Marinesco, Radovici and Draganesco [10] have observed conjugate deviation of the head and eyes, usually upward, but often to the side, which they attribute to possible involvement of the labyrinth. Bing and Schwartz [11] report three cases of upward movement, and place emphasis on the paroxysmal character and the tendency to quick recovery.

From the foregoing references, it may be seen that although the phenomenon has apparently been observed for not more than three years, it is of relatively frequent occurrence, and may be regarded as one of the later manifestations of the parkinsonian syndrome peculiar to the postencephalitic cases.

That the phenomenon is by no means uncommon is shown by the fact that since our attention was first called to the matter, less than a year and a half ago, thirteen entirely characteristic cases have come under our observation. A brief report of the cases follows.

REPORT OF CASES

Case 1.—J. V., aged 30, a French Canadian by birth, had a fever in 1921, during which he was delirious. He had headache, diplopia, right internal squint, and occasionally a sharp pain of short duration in the distribution of the first branch of the left fifth nerve. The nasal accessory sinuses were not involved. In March, 1924, he noticed that there was a tendency for the head to be drawn to the left and also that both eyes tended to deviate upward, remaining so for from a few minutes to three hours; this condition was occasionally accompanied by diplopia and rarely by nystagmoid movements to the left. Such attacks occurred several times a week. In October, 1924, when examined by one of us (C. A. M.), the right pupil was smaller than the left, and neither showed active reaction to light or in accommodation. Breathing was somewhat difficult. He easily became dyspneic. He was slow in movements. He was emotionally unstable, slow in conversation, lacked facial expression and was much retarded mentally. The blood Wassermann reaction was negative, and the spinal fluid showed four cells with an increase of albumin; the Wassermann reaction with the fluid also was negative. He had gained 55 pounds (24.9 Kg.) since he was first ill in 1921. A year after the first observation the involuntary movements of the head had disappeared and the ophthalmic pain had disappeared, but the pupillary phenomena and the upward conjugate movements of the eyes still persisted.

Case 2.—C. S., an American, aged 35, was slightly ill in 1922 with fever, headache, insomnia, failing vision and diplopia. In October, 1924, when he first came under observation, he was lethargic, still had diplopia and poor vision, had

9. Popowa, N.: Ztschr. f. d. ges. Neurol. u. Psychiat. **97**:515, 1925.

10. Marinesco, G.; Radovici, A., and Draganesco, S.: Rev. neurol. **41**:148, 1925.

11. Bing, R., and Schwartz, L.: Encéphale **23**:150, 1925.

increased in weight, and showed forced upward movements of the eyes. Wassermann reactions with both the blood and the spinal fluid were negative; there were eight cells in the fluid. The pupils showed slight reaction to light and no accommodative response. The right facial pull was greater than the left. There was slight external rotation of the right eye. While the examination was being made, both eyes were drawn to the right and upward, without any movements of the head, and the eyes remained in that position for several minutes. The neuromuscular mechanism did not show other abnormalities. Scopolamine was prescribed, without definite benefit.

CASE 3.—M. C. B., aged 21, a high school girl, unmarried, when 16 had an attack of what was supposed to be food poisoning. Later, she had a doubtful paralysis of one arm, which was attributed to hysteria. She gave up school, went to live on a farm and improved slightly. In 1924, while attempting to study stenography and typewriting, she broke down again nervously. It was then that she first noticed a peculiar "rolling" of the eyes. She did not improve. On further study, it appeared that the original attack was accompanied with fever, delirium and extreme drowsiness for about two weeks. For a year or more, she had had peculiar attacks which came on in the afternoon or evening, but never while she was in bed. The attacks consisted of a violent twitching of the eyelids and a sharp upward turning of the eyeballs; they lasted from a few seconds to a few minutes. The head was thrown back. She often had to be lifted onto the bed during the attack, but she was able to stop it, usually if she lay on the right side; it did not then recur if she lay quietly for a time thereafter. She had noticed that this condition of the eyes was intensified by embarrassment, and in general when she was more or less self-conscious or after an exciting day.

Physical examination showed the pupils to be normal in size, with reaction to light and in accommodation; the fields and fundi were normal; nystagmus was not present. Hearing was normal, with a positive Rinne test. The other cranial nerves were objectively normal. The tongue, throat and teeth did not show abnormality. The arm reflexes were equal—the elbow, wrist and biceps. She did not have ataxia. Sensation was normal. The grasp, as registered by the dynamometer, was 70 on the right and 60 on the left. The knee reflexes were equal and normal, and the achilles reflex was normal. There was no Romberg sign. Considerable tremor of the hands was present. Speech was not markedly affected, but was somewhat slow. The stiff and awkward gait, with definite lack of associated movements, was distinctly of the parkinsonian type, and the general appearance was characteristic of an encephalitic parkinsonian syndrome. The face was somewhat masklike. On several occasions, while being examined, the eyes rolled strongly upward and usually somewhat toward the right. Evidently, there was no loss of consciousness. With the spasmodic movement of the eyeballs, there was considerable tremor of the lids. After a time, the spasm relaxed and the patient became entirely normal, except for the persistent parkinsonian appearance. She was somewhat emotional at times. Treatment with scopolamine hydrobromide, beginning with 1/150 grain (0.4 mg.) three times a day, apparently resulted in some improvement, but evidently more is to be gained by attention to the general condition and avoidance of excitement and fatigue, than by the use of this or any other known drug. In general, the patient had improved somewhat when last seen, but was not well.

CASE 4.—A man, aged 30, in 1921 had fever, was delirious, and suffered from headache and diplopia with strabismus and facial pain. In 1924, he first noticed abnormal involuntary movements of the head to the left, and with these movements and occasionally without them, both eyes would turn upward, remaining so

for from a few minutes to several hours. These movements of the eyes with the turning of the head occurred several times a week and were occasionally accompanied by diplopia. At this time, in October, it was noticed that the right pupil was smaller than the left, with slight reaction to light and in accommodation. The patient developed difficulty in breathing characterized by dyspnea. He developed rigidity of the body and arms, with loss of facial expression. He became slow in conversation, mentally retarded and emotionally unstable, crying without cause. There was no evidence of disturbance of the pyramidal tracts. The Wassermann examination was negative. There were 4 cells in the spinal fluid, with an increase in protein.

The case is a clear one of epidemic encephalitis in which a parkinsonian syndrome with forced upward conjugate movements of the eyes developed. Treatment by scopolamine, 1/100 grain (0.6 mg.) twice daily, led to both general and local improvement.

Case 5.—A man, aged 35, in 1922, had fever, headache and insomnia with failing vision and diplopia. He was first seen by one of us (C. A. M.) in October, 1924, and then showed lethargy, diplopia, defective acommodation response and increased weight, with forced upward movement of the eyes. During the course of the examination both eyes were drawn slightly to the right and upward, remaining in that position for several minutes. When he assumed a reclining position with relaxation, the eyes returned to a normal position. Subsequent examination showed slight reaction of the pupils to light and none in accommodation. The right nasolabial line was more pronounced than the left; there was slight right external squint. The eyes were prominent; the gaze fixed. The patient was pale, with an emotionless facial expression and a wet mouth. He complained of a sensation of "general shaking" of the body, but there was no obvious tremor. The gait showed a tendency to propulsion, with inequality of the associated automatic movements of both arms. There were eight cells in the spinal fluid; the serology otherwise was not abnormal. The combination of encephalitic parkinsonian syndrome and abnormal forced upward movement of the eyes is again apparent. In this case, the administration of scopolamine accomplished nothing.

Case 6.—A woman, aged 23, in August, 1920, had fever, lethargy, weakness of the left external rectus and an increased right nasolabial fold, without involvement of the pyramidal tracts. In July, 1925, a diagnosis of encephalitis with parkinsonian syndrome was made by a competent physician. One of us (C. A. M.) had seen her in 1920, and again in January, 1926. At the later date, she showed a typical parkinsonian syndrome with mask face, poor control of laughter, propulsion and rigidity and loss of associated automatic movements of the arm and head. She had gained 50 pounds (22.7 Kg.). A tremor of the arms and head of the paralysis agitans type had developed, the right arm being more involved than the left. The tendon reflexes were normal. She still had diplopia and had developed a forced upward movement of the eyeballs which occurred several times a week, the eyes drawing up and remaining so for perhaps a half hour. A readjustment of the eyes was possible only, for example, by playing the piano or by reclining. During the attack, she had pronounced vertigo. The pupils reacted to light and in accommodation. At times there was a spasm of the left frontalis muscle. The spinal fluid showed normal pressure and serology. This patient improved decidedly with the administration of scopolamine in regard to both the movement of the eyes and the general condition.

Case 7.—A man, aged 34, in February, 1922, had fever, tremor, diplopia and drooling. The symptoms persisted, and the diagnosis of "sleeping sickness" was

made. He was first observed by one of us (C. A. M.) in 1926. The white blood cells numbered 11,900, with normal serology. The Wassermann test was negative, but there was persistent diplopia due to weakness of the right internal rectus, with bilateral spasm of the eyelids and forced conjugate upward movements of the eyes. In this case, the eyeballs usually remained up for several minutes, but occasionally the time was much shorter. In the attempt to force them down to a normal position spasm of the lids developed, and when the mouth was opened the eyes closed. The pupils reacted to light but not in accommodation. There was increased tone of the arms and legs, greater on the left than on the right. The tendon reflexes were brisk; there was tremor of the left hand and lack of associated automatic movements of the arms, legs and head. Here again encephalitis, with a parkinsonian syndrome, was associated with forced conjugate upward movements of the eyes. Treatment by scopolamine, 1/200 grain (0.3 mg.), four times a day and salt tablets every morning led to improvement in the general and local conditions.

Case 8.—A man, aged 35, without a history of definite infection with involvement of the cranial nerves, although he is said to have had influenza several times during the preceding eight years, developed a parkinsonian syndrome. In so young a man, this, together with ocular difficulties, places the case in the category of postencephalitic disturbances. In July, 1925, the patient went to an ophthalmologist because of difficulty in controlling an upward movement of the eyes. This occurred every four days and remained for a long time; he had found it necessary to go to bed before the eyes would assume a normal position. For several months previous to this experience, the eyes had also been drawn down at times. On one occasion, he drove forty-four miles with the eyeballs drawn down and was unable at any time to bring them to a level. At times, the pull alternated; for example, an upward pull might be followed by an attack of downward pulling. At a more recent visit he said that the attacks were becoming less frequent, but that in driving an automobile, if he saw a stone ahead in the roadway he was unable to change his gaze until the stone was passed. A rest in bed or a nap relaxed this fixity, whether it was upward or downward. In addition to these forced conjugate movements of the eyeballs, he had an oily, spastic, smiling face and a wet, tremulous tongue. There was hypertonicity of the right arm and leg with normal tendon reflexes, diminution of automatic associated movements of the right arm and tremor of this arm. He said that the loss of motor control of the right arm had caused him to become left-handed. There had been a great increase in weight. The Wassermann reaction was negative, and the spinal fluid was not examined. This case, similar in most respects to the others, differs in that it showed a forced conjugate downward movement of the eyes, as well as an upward movement associated with the usual encephalitic parkinsonian syndrome. Treatment by salt every morning and small doses of scopolamine greatly improved the hypertonicity and reduced the frequency and duration of the abnormal movement of the eyes.

Case 9.—G. A., an Italian, aged 32, unmarried, employed in an iron foundry, who was a pugilist but had not boxed since the end of 1921, had always been well until about six years previously; then, without an antecedent history of diplopia or drowsiness, the arms, body and legs became tremulous and the movements were greatly "slowed up." It became difficult for him to dress, and activity in various directions was decidedly diminished. The onset of this condition was apparently gradual; he was well in other respects. At the time we saw him, he thought that he was growing slightly worse. A year and a half previously, the eyes began to turn upward involuntarily; the head also showed a

tendency to turn backward. The eyes occasionally were turned toward the side as well as upward. The duration of the attacks of upward rotation was from nine to ten hours, rarely shorter; the eyes seemed "stuck" in that position. When he went to bed the spasm subsided, but if he would lie down during the day, the spasm returned as soon as he assumed an upright position. The difficulty increased. He had attacks from two to three times a week; they were unchanged, however, in regard to duration. He sometimes laughed involuntarily, and cried more easily than heretofore.

Examination on Dec. 15, 1926, showed the pupils to be equal, normal in size and round, with retained response to light and in accommodation, normal fields and no nystagmus. The other cranial nerves were normal, except for a masklike face. The arm reflexes were active; ataxia was not present; as measured by the dynamometer, strength was equal and normal in the two hands. The patient felt a subjective sensation of numbness in the left arm, together with considerable tremor. The knee and achilles reflexes were active, but without clonus; the plantar responses were normal and there was no disturbance of sensation or of sense of position in the legs; the abdominal reflexes were present. The patient presented a typical parkinsonian appearance in regard to facial expression, associated movements, tremor and rigidity. The combination, therefore, of a parkinsonian syndrome with spasmodic upward deviation of the eyes was again evident.

Case 10.—A. M., a woman, aged 21, unmarried, who was examined Dec. 15, 1926, had been in general well, except for enteroptosis, for which she had previously been treated. Five years previously she had become drowsy and had been ill during the night with severe diarrhea; there was no history of fever or diplopia. After recovering from this acute attack, she was drowsy for two or three months but then appeared wholly recovered. In January, 1926, the eyes began to turn up spasmodically, each attack lasting from fifteen or twenty minutes to four hours. During these attacks she was incapable of forcing the eyes downward. There was no associated diplopia. At times she was unable to close the eyes at night, and therefore could not sleep. The attacks occurred at variable intervals, usually twice a week, but at times with an interval of three weeks. At about the time of the onset of the spasmodic movements of the eyes, she noticed a beginning rigidity and "slowing up" of activities. She became emotional, laughing and crying easily and falling off in weight. Examination showed slow pupillary reaction with good accommodative response. The pupils were small, with normal fields and no nystagmus. Apart from a masklike face and slight facial asymmetry, the cranial nerves were normal; the blood pressure was slightly low; the heart was normal and the pulse rate, 88. The arm reflexes were active, and there was no ataxia; sensation was normal; the knee and achilles reflexes were active; the plantar reflexes were normal; there was no clonus, and sensation and sense of position were unaffected. The patient presented a definite parkinsonian appearance, with the usual lack of associated movements, especially on the right. She was drowsy during the examination. The combination, therefore, of the parkinsonian syndrome and upward deviation of the eyes was again present. Treatment by scopolamine led to great improvement.

Case 11.—T. F. M., aged 26, a married man, was examined by Dr. H. R. Viets on Sept. 21, 1926, who has given us the following notes: The patient had been well until the fall of 1923, when he had had a sudden onset of diplopia which had lasted about one month. He had not had fever or headache and he had not vomited, but soon after the onset of diplopia he had become drowsy. He had been obliged to give up work (driving a baker's wagon) for a month. In

spite of sleeping during the day, he would also sleep twelve hours at night. He finally gave up his work entirely, but after failing in another attempt to obtain relief from his condition, he again returned to driving the wagon. Since the onset, in 1926, he had not felt so well as formerly. He tired easily and had great difficulty in getting about. He had had some drooling at night but few other pronounced symptoms until the summer of 1926, when for the first time he began to have attacks of "rolling up of the eyes." As he sat quietly or while walking, the eyes rotated upward, the head tipped back, and he was unable to force the eyeballs into proper position for one or two minutes. He did not feel discomfort after the attacks were over. The attacks occurred as often as twice a day, but they usually averaged two or three a week. At the time of examination, he did not have diplopia. According to a friend, his personality had changed; he was more irritable and less ambitious than formerly. His habits were good. All the teeth had been removed. Physical examination showed: weight, 122 pounds (55.3 Kg.); equal pupils, the left reacting slightly to light, the right normal; the accommodative response was slight in both pupils; ocular fundi, normal; no nystagmus; other cranial nerves, normal. The arm reflexes were normal. The face was slightly masklike. There was no tremor, but the arms showed loss of associated movements.

It will be seen in this case that after a mild attack of encephalitis the sign of forced upward deviation of the eyes developed after a period of three years, which may be regarded as the usual history.

CASE 12.—K. M., aged 24, married, a housewife, about four years previously was drowsy for three or four days and had double vision which lasted about four weeks. The condition came on with fever, and a diagnosis of epidemic encephalitis (lethargica) was made. The patient had never been wholly well since; her capacity for work had been limited, but she had been able to do ordinary housework. She had been married two years previously, and had a child, aged 15 months. No special difficulty was experienced at the birth of the infant. She had had a history of rheumatism previous to this illness, but in general had been ordinarily well. Three or four months previously, while she was eating supper, the eyes suddenly turned upward and the head was forced back. This lasted only a few minutes but recurred thereafter approximately every other day. The only way that these attacks could be relieved was by lying down. There was no twitching of the eyelids. Since her first visit to the Massachusetts General Hospital on Dec. 17, 1926, she had continuously taken scopolamine hydrobromide, beginning with 1/200 grain (0.3 mg.) two or three times a day. The dosage was increased to 1/200 grain three times a day, and she was decidedly better in all respects. The attacks were not so frequent or so severe as formerly, and the general rigidity had been lessened. Examination showed a typical slight parkinsonian condition. The face was masklike; the movements were slow; the gait was somewhat, but not markedly, characteristic of the paralysis agitans syndrome. There was considerable lack of associated movement in the arms. Physical examination did not show any objective disturbance of the cranial nerves beyond the facial rigidity. The arm reflexes were normal; the knee reflexes were also within normal limits, and the achilles response was present. The Romberg sign was not elicited, and no objective disturbance of sensation was evident. There was a possibility of slight difficulty in convergence and in accommodative response; otherwise the eyes were normal. The Wassermann reaction was negative.

In this case, the outstanding result of the encephalitis was the common paralysis agitans syndrome and the pronounced upward deviation of the eyes, corresponding closely to the usual type.

CASE 13.—F. L., a man, aged 21, a patient at Long Island Hospital, unmarried, a former worker at a piano factory, was well until four years previously, when he had had an attack of fever and drowsiness, and had been ill for about two weeks. He had never fully recovered from this attack, but was able to work on occasional jobs. About two years previously, he began to show definite signs of parkinsonism. It became hard for him to get about, to dress himself and to attend to ordinary needs. The onset was gradual. In April, 1925, and therefore not long after the beginning of the parkinsonian syndrome, while he was eating his evening meal, the eyes suddenly turned upward and were held fixed in that position. This lasted three hours, and was relieved only by lying down, and then not immediately. Since that time, he had had such attacks approximately once in four days, varying in length from perhaps three minutes to three or four hours. In the attacks the head was drawn backward, apparently compensatory to the deviation of the eyes. One such attack was observed as he came into a room to be shown at a clinical meeting. Suddenly the eyes were strained involuntarily upward, possibly a little to the side; the head was elevated but not drawn violently backward. He continued to walk about, evidently in considerable discomfort; he finally sat down, put a hand over his eyes, made various muscular movements, and the lids trembled; then, as suddenly as the seizure began, there was complete relaxation and the eyes resumed a normal position. The spasm was not unlike that seen occasionally in an ordinary epileptic attack, as far as the eyes were concerned, except for its long duration.

The man was well built, with no obvious defect except the parkinsonism, so far as the general condition was concerned. The pupils were unequal, the right larger than the left, and were fairly circular in outline. The reaction to light was extremely slight in the right eye, though it was decidedly better in the left. The accommodative response was poor and convergence defective. The fields in general were normal. There was extremely slight lateral nystagmus. Hearing was unaffected. The face was masklike, with more mobility on the left than on the right. The other cranial nerves were unaffected. The tongue was tremulous and clean; the teeth were in fair condition. The arm reflexes were all active but within normal limits. There was no ataxia, but slight tremor developed on intended movement. The leg reflexes were unequal; the knee and achilles reflexes both were active. There was no clonus or plantar response, but a strong suggestion was present of a Babinski response on the left. The Romberg sign was not present. Sensation and position sense in the legs were normal. The heart was normal; the pulse rate was 116 during the examination. The systolic blood pressure was 120; the diastolic, 80. The abdominal reflexes were present and the sphincters unaffected. The gait was typical of the parkinsonian syndrome, the right side being distinctly more rigid than the left, with lack of associated movement in that arm. Speech was characteristically slow and monotonous. The urine was normal; both the blood and the spinal fluid gave negative Wassermann reactions. The various constituents of the spinal fluid were not noteworthy.

This case is another instance of an unquestioned original encephalitis, followed in about two years by a parkinsonian syndrome, with the development of upward deviation of the eyes several months after the onset of the parkinsonian rigidity. The patient was greatly helped by scopolamine.

COMMENT

A general analysis of the foregoing cases emphasizes the following facts: The movements of the eyes in all cases were predominantly upward; in eight instances, this was the only movement described; in four this was combined with lateral movement; in one, there was slight external rotation of the right eye, and in one (case 8) downward alternated with upward movement. Associated movements of the head occurred in five cases, and in some of these they appeared to be semivoluntary to supplement the strained position of the eyes. The frequency of the attacks varied widely from those that occurred daily to those that occurred two or three times a week; in general, the intervals were more than twenty-four hours. Furthermore, there was no constant interval between the attacks. In one instance, fatigue and embarrassment seemed to increase the frequency. The duration of the individual seizures was likewise most variable, from one or two minutes to nine or ten hours. Between these extremes were attacks that occurred at intervals of half an hour or a few hours, again without constancy of time in individual cases. Other accompanying conditions of the eyes were presumably of little significance in relation to the special phenomenon of deviation; for example, diplopia, defective light and accommodative response, twitching of the eyelids and spasm of the frontalis muscle. The treatment, largely by scopolamine, was definitely effective in eight cases; in two, it was without result. The disappearance of the spasm when the patient lay down was striking in several of our cases, as it was in others reported. At times this was the only way in which the spasm could be overcome. One patient was obliged to lie on the right side to accomplish the desired result. In one instance (case 10), however, the opposite occurred; the patient at times was unable, because of the spasm, to close the eyes at night and was thereby prevented from sleeping.

In the absence of postmortem examination, it is fruitless to speculate as to the character and exact location of the lesion producing the movements, apart from the doubtless justified assumption that it lies in the region of the striatum or in the coordinating mechanism of the ocular nerves. The possibilities have been exhaustively discussed, but without unanimity of opinion, in the various articles to which reference has been made. That the lesion occurs only in postencephalitic parkinsonism appears well established, and the inference is therefore plausible that it is due to the same lesions that underlie that syndrome. At present, further assumptions are not justified.

THE OCULO-AURICULAR ASSOCIATED MOVEMENT

ITS POSSIBLE UTILITY AS A TEST OF THE FUNCTION OF THE FACIAL NERVE *

RICHARD M. BRICKNER, M.D.
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BALTIMORE

A facial associated movement of considerable interest was discovered by Wilson [1] in 1908. He found that when the eyes are deviated extremely to one side or the other, there is a slow contraction of one of the intrinsic muscles of the ear, the musculus transversus auris. This small muscle, situated on the posterior aspect of the concha, spanning the fossa antihelicis, and connecting the helix and the antihelix, draws the pinna posteriorly. The muscle fibers are supplied by the seventh cranial nerve. The contraction is maintained as long as the eyes remain in the position of lateral gaze. Wilson examined fifty normal men in London, and found the movement to be present in only 40 per cent. The details of his observations are reviewed in table 1. Within the same year Stannus,[2] a British army surgeon, recognizing that the phenomenon appeared to be the vestige of some ancient function, conceived the idea that primitive man might show the oculo-aural movement, as he termed it, in larger proportions than Wilson's group. Accordingly, he examined 100 healthy males, belonging to several tribes of the Bantu family in Nyasaland, Central Africa, and found that 66 per cent of them exhibited the movement in one or both pinnae. The literature apparently contains no additional comments on the phenomenon until 1924, when Carmichael and Critchley [3] examined 400 men and women in London, observing the action in 50 per cent.

The present reports covers an investigation of the oculo-aural responses of fifty white men, fifty white women, fifty colored men and fifty colored women, attending the Johns Hopkins Hospital dispensary. None of the persons was known to be suffering from a neurologic ailment, and lesions of the cranial nerves were sought and excluded, within the range of gross tests, in all of them.

For the observation of the aural reaction, the subject should stand squarely facing a good light, the examiner being either directly in front of or behind him, or slightly to one side. The movement is most prominent at the upper part of the pinna, where the motion

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* From the Medical Clinic, Johns Hopkins Hospital.

1. Wilson, S. A. K.: Rev. Neurol. & Psychiat. **6**:331, 1908.
2. Stannus, H. S.: Rev. Neurol. & Psychiat. **7**:770, 1909.
3. Carmichael and Critchley: J. Neurol. & Psychopath. **5**:124, 1924-1925.

is exaggerated by the greater helical breadth. Usually, the movement is best seen from the front, but sometimes, especially when it is slight, it can be seen to best advantage from the posterior aspect. The eyes should be rotated, as swiftly and steadily as possible, to the most extreme point in the given direction.

The results of the test are summarized in table 1.

An aural response to upward or downward motion of the eyes is less frequently found than to lateral motion, as table 1 shows. Such reactions, when present, are usually typical, though less extensive. However, one subject (M. F., aged 19) showed a pronounced upward or downward movement of the entire ear against the head, accompanying the corresponding ocular deviation.

The present series gives a much higher ratio of positive tests than any hitherto reported. The response is found more often among the whites than among the Bantus of Stannus, while the proportion of positive responses among the negroes is within the range of his figures. In order to attain an accurate conception of the movement as it exists in more and less primitive races, it is clear that additional and numerous inquiries are needed.

In the present study, as the table shows, the maximal movements were always equal in range on the two sides, or approximately so, except in seven cases. Two of these displayed a facial weakness on the side giving the smaller range of motion; in one, there was a history of facial neuralgia on the side with the greater range, a disturbance that may possibly have been concerned in producing the inequality, and in one the corresponding side of the face was badly scarred from an injury. The three remaining cases, all in colored people, were unexplained. Inequalities in the extent of the minimal movements were more common. The previous authors have reported larger numbers of inequalities between the motions of the two ears; whether the discrepancies were related to the minimal or maximal movements is not stated, however.

The nature of the phenomenon has puzzled all of its students. Wilson termed it an "associated movement." Carmichael and Critchley considered, too, that it is an "associated movement"; that it is not a reflex is evidenced, they thought, by its selective character, its slowness, and its absence in the majority of their cases. All have inferred that it probably represents a vestigial function.

Believing that some light might eventually be thrown on the basis of the reaction through knowing whether or not it belonged among the so-called automatic associated movements that are lost in paralysis agitans, I examined eight patients having that disease at the Montefiore Hospital, New York. All of the patients showed the movement. The difficulties besetting the testing of such individuals are manifest; the

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TABLE 1.—*Results of Investigations of Oculo-Aural Response*

	Present	Present (But Slight)	Absent	Contralateral Motion Greater Than Homolateral	Contralateral Motion Equal to Homolateral	Homolateral Motion Greater Than Contralateral	Homolateral Motion Absent	Contralateral Motion Absent	Eyes Turned Down R = L	Eyes Turned Down (Response by One Ear Only)	Eyes Turned Up R = L	Eyes Turned Up (Response by One Ear Only)	Maximal Movements R = L	Maximal Movements (Possibly Very Slight Inequality)	Maximal Movements (Marked Inequality, Unexplained)	Maximal Movements (Marked Inequality Explained)	Special Points
Wilson																	
(50 men).....	20 40%	..	30 60%	5 (both ears) 1 (one ear only reacting)	..	2 (both ears) 5 (one ear only reacting 6 (no contra.)	1	11	..	..	..	..	..	..	9	..	One case in which the range was greater homolaterally in one ear and contralaterally in the other, though maximal movements were equal
Stannus																	
(100 Bantu men)	66 66%	..	44 44%	79%*	19.7%	1%	..	..	..	..	..	..	..	..	11	..	0
Present Series																	
White men.....	44	4	6	35	7	2	1	1	38	1	8	2	44	0	0	0	0
(50)	88%	9%	12%	79%	16%	4%	2%	2%	86%	2%	18%	4%	100%				
White women.....	41	4	9	32	4	4	10	3	5	2	4	0	39	2	0	0	(1) One case in which the range was greater homolaterally in one ear and contralaterally in the other, though maximal movements were equal. (2) one case in which the whole ear moved up and down against the head as the eyes were moved upward or downward
(50)	82%	9%	18%	78%	9%	9%	24%	7%	12%	5%	9%		95%	5%			
Colored men.....	35	1	15	23	2	6	8	5	3	0	2	1	30	2	2	1	One case in which the range was greater homolaterally in one ear and contralaterally in the other, though maximal movements were equal
(50)	70%	3%	30%	65%	5%	17%	22%	14%	8%	0	5%	3%	86%	5%	6%	3%	
Colored women...	37	3	13	31	1	2	10	1	13	0	3	0	31	2	1	3	0
(50)	74%	8%	26%	83%	3%	5%	27%	3%	35%	0	8%		84%	5%	3%	8%	

* These percentages refer to the total number of active pinnae.

eyes are moved slowly and unevenly and often not to the extreme point; therefore, no attempt was made to gain information about homolaterality and contralaterality. The only difference noted between the responses of these and normal people was a possible increase in the normal slowness of the motion. It is probable, therefore, that the ear extension is not of that group of automatic associated movements supposedly mediated through the corpus striatum and the substantia nigra.

The significance of the movement in lower animals has brought forth considerable debate. Wilson, in whose cases the reaction was most pronounced in the homolateral ear, suggested that a helpful widening of the external auditory meatus was produced on the side toward which auditory attention was directed. However, the observations of Stannus and of Carmichael and Critchley, as well as my own, concur in demonstrating that the contralateral ear is usually the one mainly affected. In man, when increased audition is desired, the aim is not to widen the external canal, but to increase the size of the concha by placing the hand appropriately behind it. Conversely, when sounds approach from behind, or from the opposite side and behind, the concha, perhaps, interferes with the entrance of sound waves into the auditory channel. Since the eyes must be turned laterally to an extreme point, implying simultaneous rotation of the head, in order to elicit the aural response, it is probable that the sounds which bring the mechanism into play are usually placed not only to one side of the animal, but also somewhat behind. Retraction of the concha toward the head, on the side away from the sound, might well aid audition under those circumstances. Such, possibly, is the function of the oculo-aural phenomenon, at least in the primates, whose ears are laterally situated. In other types of ear, such as those of dogs, it is almost impossible to discover the analog of the movement, since the entire structure of the external ear differs so radically from that of the primates.

THE POSSIBLE UTILITY OF THE OCULO-AURAL PHENOMENON AS A TEST OF FUNCTION OF THE FACIAL NERVE

It is my purpose to suggest the possibility of using the oculo-aural phenomenon as a test for the functional capacity of the facial nerve. Instances are frequently encountered, exhibiting suggestive flattening of one side of the face, in which it is difficult, if not impossible to learn the real status of the seventh nerve by means of the ordinary tests. There are also cases in which a pronounced facial asymmetry suggests a palsy of the seventh nerve, which is not present. The aural response to the ocular rotation is definite, involuntary, easily observed and, nearly always in my experience, the maximal reactions of normal white people are either equal in range on the two sides or are so nearly equal that

any difference is doubtful. On the contrary, there is a well marked diminution in the extent of the helical retraction when a definite impairment of the facial nerve exists.

Nine such cases have been observed in the neurologic clinic at the Johns Hopkins Hospital, and on the neurologic service at the Bay View Hospital, Baltimore. The results are summarized in table 2.

I have seen two patients at the Bay View Hospital who presented what seemed to be an extremely slight facial weakness, but in whom the changes were so minute that a positive diagnosis of palsy of the facial nerve could not be made on the facial symptoms alone. But the fact that both of the men had hemiplegia, coupled with slight unilateral facial flattening and drooping of the lips, and a slight lagging of the lips on that side during retraction of the lips, made the diagnosis of

TABLE 2.—*Observations in Cases of Facial Palsy*

Type of Case	Number of Cases	Maximal Movement in Ear of Normal Side	Maximal Movement in Ear of Paralyzed or Weak Side
Bell's palsy	2	Pronounced	None
Left facial paralysis due to fall on ear	1	Pronounced	None
Hemiplegia	5	Pronounced	Very slight
Hemiplegia	1	Pronounced	None

TABLE 3

	Maximal Movement in Ear of Normal Side	Maximal Movement in Ear of Side Suspected of Facial Palsy
Case 1, O. B.	Pronounced	Very slight
Case 2, G. M.	Pronounced	Very slight

unilateral facial nerve palsy the presumptive one. One of the patients gave a history of facial swelling accompanying his apoplectic stroke ten years previously. In both of these instances there was a decisive difference between the ranges of retraction of the right and left helices, on rotation of the eyes. As table 3 shows, the helix of the side suspected of facial weakness showed a definitely smaller range of movement than its fellow.

That the difference between the movements of the ears is more striking than that between other facial motions under the condition of slight palsy of the facial nerve is probably due to the fact that the movement of the ears is normally small in scope and is a more delicate indicator of slight diminutions than are the grosser ones.

ABSTRACTS OF PROTOCOLS OF PATIENTS IN TABLE 3

CASE 1.—*History.*—O. B., aged 64, a white American laborer, who was well nourished, presented a flaccid paralysis of the right arm and right leg. A slight

weakness of the left side of the face was suggested by slight flattening and a slightly greater pull on the right side during retraction of the lips and on opening the mouth widely. The tongue deviated to the left, however. Speech was thick. The deep reflexes were hyperactive on the right, and were normal on the left. Other neurologic tests give normal results. The blood Wassermann reaction was negative. The systolic blood pressure was 204; diastolic, 110.

Course.—Function of the arm and leg had improved little, but speech had improved considerably.

Diagnosis.—The condition was diagnosed as arteriosclerosis, hypertension and cerebral hemorrhage. Probably the lesion involved the left pyramidal tract and the left seventh and twelfth nerves.

CASE 2.—G. M., aged 71, a white American farmer, presented a spastic paralysis of the left arm and leg. Slight weakness of the left side of the face was indicated by slight flattening of the region of the left lower eyelid and of the left nasolabial fold. The hemiplegia was of nine years' standing. There was also an area of numbness, tingling, redness and slight tenderness involving the right little and ring fingers and the ulnar edge of the right side.

The Wassermann reactions of the blood and spinal fluid were negative. The spinal fluid was clear, with 2 cells, and the Pandy reaction negative. The systolic blood pressure was 205; diastolic, 110.

The test would be applicable only to persons in whom the ear of the normal side gave an undeniable reaction: these, as table 1 shows, average about 80 per cent of white persons. In view of the three cases of unexplained inequality that occurred among 100 normal negroes, it would be best to restrict the test to white people until more information is gained.

SUMMARY

1. Two hundred persons have been examined in a study of the oculo-aural phenomenon of Wilson and Stannus. Its frequency has been tabulated and its significance discussed.

2. It has been found that the oculo-aural response is present in cases of paralysis agitans. Therefore, it is unlikely that the reaction is one of the automatic associated movements supposedly mediated through the corpus striatum and the substantia nigra.

3. A striking diminution is demonstrated in the range of the movement in the presence of weakness of the facial nerve of even slight degree. The possibility of using the phenomenon in determining the presence or absence of lesions of the facial nerve in doubtful cases is discussed.

I wish to thank Dr. Ford, of the Neurologic Clinic, for his assistance, Doctors Boas, Kraus and Silverman of the Montefiore Hospital and Doctors Hooper Smith and Humphreys of the Bay View Hospital for their courtesies.

THE ERYTHROCYTE SEDIMENTATION REACTION

ITS VALUE IN PSYCHIATRY *

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By erythrocyte sedimentation reaction is meant the speed with which the red blood cells settle in a column of citrated blood. Many authors credit Galen with being the first to describe this phenomenon under the name of crusta phlogistica. In 1791, John Hunter studied the blood sedimentation reaction:

> He not only observed that the erythrocytes, during an inflammatory process, settled more quickly in their own plasma, but he was also the first to demonstrate that the red blood cells of normal blood, when separated and transferred to the plasma of inflammatory blood, settled with greater speed, the rapidity of the process being in direct relation to the severity of the infection.[1]

From 1894 to 1897, Professor Biernacki [2] of Warsaw published several papers bringing out the clinical value of the sedimentation test. His works were later confirmed by Müller [3] in 1898. For the next twenty years, little attention was given to this phenomenon. It is only since the work of Fahraeus,[4] in 1918, on the blood of pregnant women that so much interest has been given to the erythrocyte reaction. Hundreds of articles have been written on this subject in almost all fields of medicine. It has been the consensus of opinion in practically all cases that the test is of clinical value.

In obstetric and gynecologic studies, the sedimentation test has been extensively used. Friedlaender [1] and others assert that it is of value in differentiating pregnancy, after the fourth month, from benign pelvic tumors. Falta, Rumpf, Pewny and Hallberg believe that the test is of greater value than the temperature curve or the leukocyte count. They also have found the test helpful in determining the advisability and time for operative intervention. Baer and Reis [5] believe the test of practical value in many gynecologic conditions. Noyes and Corvese [6] have

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* From the Psychiatric Service of the Worcester State Hospital.

1. Friedlaender, B.: Blood Sedimentation Test as Aid in Diagnosis in Surgical Infections, Am. J. Obst. & Gynec. **7**:125, 1924.

2. Biernacki, E.: Ztschr. f. physiol. Chem., 1894, vol. 19; Gaz. lek. **17**:962, 1897; Deutsche med. Wchnschr. **23**:769, 1897; Ztschr. f. physiol. Chem. **23**:368, 1897.

3. Müller, G. F. O.: Beobachtungen über spontane Blutsedimentierung, Berlin, 1898.

4. Fahraeus, R.: Ueber die Ursachen der verminderten Suspensionsstabilität der Blutkörperchen während der Schwangerschaft, Biochem. Ztschr. **89**:355, 1918.

5. Baer and Reis: Sedimentation Test in Obstetrics and Gynecology, Surg. Gynec. Obst. **40**:691, 1925.

6. Noyes and Corvese: Significance of Blood Sedimentation Time in Gynecology and Obstetrics, Boston M. & S. J. **195**:891 (Nov.) 1926.

found that the sedimentation test is sufficiently dependable to warrant its use in gynecology in all cases in which a leukocyte count is considered desirable.

In surgical investigations, much has been done on the sedimentation reaction. Löhr,[7] Gragert,[8] Haller[9] and others believe that it is of decided significance in surgical diagnosis, and that it offers a reliable indication of the extensiveness and acuteness of any inflammatory lesion; they feel that it is of prognostic value and is helpful in determining the time of surgical intervention. They also have found the sedimentation phenomenon useful in distinguishing benign from malignant growths. Rubin[10] feels that the test is a more reliable indication of the condition of the patient than the temperature curve, and that it is helpful in determining the time of operation.

In the field of general medicine, in neurologic conditions and in tuberculosis, especially the latter, the test has been found of clinical value. Morriss,[11] Morriss and Rubin[12] and others believe it to be of great significance as an indicator of the degree of activity present. Cutler[13] feels that it is more helpful than the temperature, pulse or weight curve.

Comparatively speaking, little has been done with the sedimentation reaction in psychiatry. Plaut[14] found that the erythrocyte sedimentation reaction was quicker in general paralysis and psychoses due to arteriosclerosis than in the majority of cases of psychopathic personalities, hysteria, melancholia, dementia praecox and epilepsy. Runge[15] reports an increase in general paralysis, tabes, syphilis of the brain, arteriosclerotic psychoses, inflammatory cerebral processes, and also in

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7. Löhr, W.: Die Senkungsgeschwindigkeit der roten Blutkörperchen als diagnostisches Hilfsmittel bei chirurgischen Erkrankungen, Mitt. a. d. Grenzgeb. d. Med. u. Chir. **34**:229, 1921.

8. Gragert, O.: Die Bedeuntung der Senkungsgeschwindigkeit der Erythrocyten für die Diagnostik des Carcinoms und für die Feststellung der Rezidiofreiheit nach operativer Behandlung Arch. f. Gynack. **118**:421, 1923.

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10. Rubin, E. H.: The Clinical Value of the Erythrocyte Sedimentation Reaction in Surgery, Surg. Gynec. Obst. **42**:652 (May) 1926.

11. Morriss, W. H.: The Value of Erythrocyte Sedimentation Determinations in Pulmonary Tuberculosis, Am. Rev. Tuberc. **10**:431, 1924.

12. Morriss, W. H., and Rubin, E. H.: The Sedimentation Reaction of Erythrocytes. Clinical Applications and a Micromethod, J. Lab. & Clin. Med. **11**:1045 (Aug.) 1926.

13. Cutler, J.: The Graphic Presentation of the Blood Sedimentation Test: A Study in Pulmonary Tuberculosis, Am. J. M. Sc. **171**:882 (June) 1926.

14. Plaut, F.: Sinking Velocity of Blood Corpuscles in Nervous and Mental Diseases, München. med. Wchnschr. **67**:279, 1920.

15. Runge, W.: München. med. Wchnschr. **67**:953, 1920.

many cases of dementia praecox. Paulian and Tomovici [16] did not obtain a single negative result in general paralysis. In a later work, Demètre and Tomovici [17] obtained similar results. In 1924, Glaus [18] observed that there was an increase in the speed of sedimentation in senile dementia, neurosyphilis, general paralysis, catatonia and in the period immediately following an epileptic seizure. On the other hand, the reaction was less in constitutional psychopathies, neurasthenia, hysteria, manic-depressive psychoses and paranoia. According to Jacobowsky,[19] the speed of sedimentation varies directly with the amount of organic deterioration present.

TECHNIC

The technic I have used is a modified Westergern method, as suggested by Morriss.[11] A 3.8 per cent solution of sodium citrate is drawn up to the 0.4 cc. mark of a sterile 2 cc. record syringe. A venipuncture is then done, and the blood is aspirated to the 2 cc. mark. The mixture is transferred to a small Wassermann test tube and is slowly but thoroughly mixed once. The blood is then drawn up into a long 1 cc. serologic pipet graduated into 100 parts.

This is placed in a specially constructed rack. Readings are then taken directly in percentages at the end of the first and second hours. The twenty-four hour readings are not important.

TABLE 1.—*Readings in Twenty-Five Normal Cases*

	Age	Sex *	Sedimentation Per Cent 1 Hr.	Sedimentation Per Cent 2 Hrs.		Age	Sex *	Sedimentation Per Cent 1 Hr.	Sedimentation Per Cent 2 Hrs.
1	23	♂	1	2	14	39	♂	3	7
2	19	♂	1	2	15	32	♀	3	6
3	22	♂	1	2	16	40	♀	3	6
4	31	♂	1	2	17	32	♀	3	6
5	19	♂	1	2	18	30	♀	4	7
6	21	♂	2	4	19	28	♀	3	7
7	22	♂	2	4	20	22	♀	4	8
8	24	♂	2	5	21	19	♀	4	9
9	32	♂	2	5	22	20	♀	4	9
10	19	♂	2	5	23	29	♀	5	10
11	20	♂	3	5	24	26	♀	5	10.5
12	22	♂	3	6	25	25	♀	5	11
13	47	♂	3	7					

* In this table and in tables 5, 6, 7, 8, 9 and 10, ♂ indicates male; ♀, female.

RESULTS

A series of twenty-five normal persons was studied, and the results are shown in table 1.

16. Paulian and Tomovici: The Sedimentation Reaction in Nervous Diseases, Klin. Wchnschr. **2**:2174, 1923.

17. Demètre and Tomovici: Le phénomène de la sédimentation dans les affections du systéme nerveux, Paris méd. **13**:234, 1923.

18. Glaus: Sedimentation Test in Psychiatry, Schweiz. med. Wchnschr. **54**: 260, 1924.

19. Jacobowsky: Sedimentation Speed of Erythrocytes in Dementia Praecox, Upsala Läkaref. Förh. **30**:227, 1924.

TABLE 2.—*Sedimentation Reactions in a Group of Males Having Dementia Praecox*

No.	Age	Clinical Diagnosis and Condition *	Date	Sedimentation, Per Cent 1 Hr.	Sedimentation, Per Cent 2 Hrs.
26	38	Acute catatonic excitement	2/20/27	4	9
		Marked mental improvement	3/ 3/27	0	1
27	31	Mixed type; patient ready for visit	1/13/27	0	1
		Patient excited and destructive	3/ 4/27	4	9
28	19	Simple type	3/ 3/27	1	2
29	50	Mixed type	1/20/27	1	2
30	17	Simple type	1/13/27	0.5	2
31	22	Simple type	1/13/27	0.5	2
32	23	Simple type	3/14/27	1	2
33	50	Type undetermined	1/11/27	2	3
34	19	Simple type	4/ 4/27	1	3
35	22	Type undetermined	3/ 3/27	1	3
36	57	Mixed type with marked mental deterioration	1/20/27	2	4
37	29	Hebephrenic type	1/20/27	1	3
38	42	Type undetermined	1/20/27	1	3
39	36	Simple type; chronic rheumatic cardiovalvular disease present	3/14/27	1	3
40	39	Type undetermined	1/15/27	1	3
41	31	Hebephrenic type	1/29/27	1	4
42	31	Simple type	4/ 4/27	1	4
43	27	Hebephrenic type	1/29/27	1	4
44	42	Mixed type; second attack	1/20/27	1	4
45	50	Paranoid type	1/20/27	1	4
46	47	Paranoid type	1/15/27	1	4
47	32	Mixed type; second attack	3/ 7/27	2	4
48	33	Simple type	3/ 7/27	2	4
49	51	Mixed type	1/15/27	2	4
50	31	Catatonic stupor; third attack	3/27/27	3	5
51	43	Hebephrenic type	1/20/27	3	5
52	32	Simple type, with mental deterioration	1/20/27	3	5
53	36	Paranoid type	1/15/27	1	5
54	26	Mixed type with marked deterioration	1/13/27	3	6
55	51	Mixed type with marked deterioration	1/15/27	3	6
56	42	Mixed type with marked deterioration	1/15/27	3	6
57	21	Hebephrenic type with marked deterioration	1/15/27	3	6
58	32	Mixed type with marked mental deterioration	1/20/27	2	6
59	50	Paranoid type; patient in hospital twelve years	1/29/27	2	6
60	54	Paranoid type; patient in hospital sixteen years	1/29/27	3	6
61	38	Simple type; second admission	3/21/27	3	7
62	26	Simple type; inguinal hernia present	3/14/27	2	7
63	50	Mixed type, with marked mental deterioration	1/15/27	2	7
64	48	Mixed type; patient in hospital six years	1/29/27	3	7
65	23	Simple type; temperature, 99; pulse, 82; respiration, 24; patient had a slight cold	3/ 3/27	3	7
		Two weeks later	3/17/27	2	4
66	34	Simple type, with marked metal deterioration	3/ 7/27	2	8
67	21	Paranoid type; tuberculous osteomyelitis of the left ankle	1/13/27	2	8
68	37	Mixed type with marked mental deterioration	1/13/27	3	8
69	28	Type undetermined; patient in hospital four years	1/20/27	3	8
70	48	Mixed type; marked mental deterioration	1/27/27	3	8
71	47	Mixed type with marked deterioration	1/20/27	3	8
72	22	Catatonic excitement	4/ 4/27	4	9
73	26	Simple type	1/13/27	3	9
74	28	Mixed type, with mental deterioration	1/20/27	3	9
75	20	Catatonic type	1/20/27	3	9
76	26	Catatonic type, with marked mental deterioration	3/ 2/27	4	9
77	33	Hebephrenic type, with marked deterioration	1/29/27	4	10
78	41	Mixed type; marked deterioration	1/29/27	5	10
79	41	Mixed type; marked mental deterioration	1/13/27	5	10
80	32	Type undetermined; patient in a state of hyperactivity	3/28/27	6	11
81	60	Mixed type; patient in hospital twenty years, has marked mental deterioration	1/13/27	4	13
82	68	Mixed type; patient in hospital fourteen years, shows marked mental deterioration	1/13/27	5	14

* In this and in the following tables, the urinalysis, Wassermann reaction, blood picture, temperature, pulse and respiration rate were all essentially normal unless otherwise stated. No physical complications were present unless otherwise stated.

The readings recorded in table 1 are more or less in agreement with those obtained by Morriss and Rubin. Readings up to 6 per cent for males and 11 per cent for females, at the end of the second hour, may be

TABLE 3.—*Readings in a Group of Females Having Dementia Praecox*

No.	Age	Clinical Diagnosis and Condition	Date	Sedimentation, Per Cent 1 Hr.	Sedimentation, Per Cent 2 Hrs.
83	32	Paranoid type	9/20/23	2	3
84	39	Simple type	8/19/26	2	4
85	49	Type undetermined	8/18/26	2	5
86	31	Paranoid type	9/ 3/26	4	7
87	29	Simple type	8/19/26	4	7
88	31	Mixed type	8/19/26	3	7
89	32	Simple type	3/21/27	2	7
90	17	Simple type	9/ 8/23	2	8
91	31	Mixed type	9/20/26	4	8
92	32	Catatonic stupor	4/ 4/27	3	8
93	47	Paranoid type	3/18/27	3	9
94	35	Simple type	8/18/26	3	9
95	29	Mixed type	8/18/26	4	9
96	44	Mixed type	9/30/26	3	9
97	47	Type undetermined	9/ 2/26	4	10
98	46	Paranoid type, with marked mental deterioration	9/ 8/26	4	10
99	31	Mixed type; marked mental deterioration	9/ 3/26	4	10
100	38	Paranoid type	4/ 4/27	3	11
101	33	Catatonic excitement	3/ 2/27	4	11
102	40	Catatonic excitement; patient here four years	10/ 1/26	5	12
103	30	Mixed type; patient's third attack	3/ 2/27	5	12
104	18	Simple type	9/20/26	3	8
		Patient much worse mentally	10/23/26	5	14
		Patient showed a great mental improvement	3/ 3/27	4	10
105	48	Mixed type; fifth attack; much mental deterioration present	3/ 2/27	6	16
106	35	Simple type; chronic interstitial nephritis	9/ 7/26	7	16
107	46	Paranoid condition	10/ 1/26	6	15
108	56	Mixed type	10/13/26	5	15
109	41	Paranoid type; history of many rheumatic attacks; chronic rheumatic cardiovalvular disease	9/ 2/26	7	17
110	20	Simple type; patient feeling well	9/30/26	4	20
		Two days later patient was sent to medical service for subacute salpingitis, gonorrheal in origin.	10/ 3/26	7	28
111	16	Simple type; patient feeling well; temperature, pulse and respiration normal	9/ 3/26	6	21
		Two weeks later, patient sick; pain and a small mass in the left lower quadrant, which, at operation was found to be a gonorrheal pelvic abcess; temperature, 101; pulse, 92; respiration, 24	10/ 1/26	20	38
112	54	Paranoid type; impacted fracture of left radius, lower end	9/ 2/26	9	25
113	30	Catatonic excitement; history of gallbladder disturbances; temperature, 99.8; pulse, 84; respiration, 24	3/ 7/27	13	29
		Two weeks later, patient greatly improved mentally	3/21/27	6	15
114	35	Paranoid type; bilateral pulmonary tuberculosis; temperature, 99.2; pulse, 84; respiration, 22	9/ 3/26	12	27
115	59	Simple type; poorly nourished and poorly developed; a large mass in the abdomen, possibly malignant; red blood cells, 3,880,000; hemoglobin, 60 per cent; white blood cells, 5,850	9/20/26	41	61
116	40	Paranoid type; hypernephroma of right kidney; red blood cells, 3,710,000; white blood cells, 6,250; hemoglobin, 40 per cent; polymorphonuclear cells, 65; lymphocytes, 27; slight anisocytosis; patient made no complaints	8/27/27	51	66
117	50	Paranoid type; malignant disease of right lung, with possible origin in mediastinum; patient made no complaints; red blood cells, 3,800,000; white blood cells, 6,700; hemoglobin, 60 per cent; temperature, 101; pulse, 90; respiration, 30	8/19/26	14	26
		Patient much worse; temperature, 103; pulse, 90; respiration 36; the patient died a few weeks later.	10/ 4/26	18	38

said to be normal; 6 and 7 per cent in males and 11 and 12 per cent in females may be considered as high normal readings.

Tables 2 and 3 give the readings in ninety-two cases of dementia praecox in both males and females. They show that the sedimentation

reaction in dementia praecox is either normal or slightly increased unless complicated by some definite physical condition. The cases in which the readings are within normal limits are, with a few exceptions, of short duration and usually the ones belonging either to the simple or paranoid types of dementia praecox. On the other hand, increased readings are found in the hebephrenic and catatonic types, more especially in the latter. The more mental deterioration present, the more accelerated is the reaction. Cases 26, 27, 104 and 113 are interesting because they show how the sedimentation reaction varies with the mental changes of

TABLE 4.—*Readings in Cases of Alcoholic Psychoses in Men*

No.	Age	Clinical Diagnosis and Condition	Date	Sedimentation, Per Cent	
				1 Hr.	2 Hrs.
118	40	Chronic alcoholic psychosis; one year duration.....	1/11/27	1	2
119	42	Chronic alcoholic psychosis; five years' duration....	1/20/27	1	3
120	38	Chronic alcoholic psychosis; three years' duration...	1/13/27	1	5
121	38	Chronic alcoholic psychosis; four years' duration...	1/20/27	2	5
122	44	Chronic alcoholic psychosis; with marked mental deterioration	1/13/27	2	5
123	42	Chronic alcoholic psychosis; six months' duration..	1/15/27	3	6
124	29	Chronic alcoholic psychosis; eight months' duration.	3/ 7/27	2	7
125	37	Chronic alcoholic psychosis; three months' duration	3/21/27	3	8
		Patient mentally worse	3/28/27	5	10
126	37	Chronic alcoholic psychosis; four years' duration ...	1/20/27	3	8
127	36	Chronic alcoholic psychosis; left knee ankylosed old tuberculous process.....	1/11/27	4	11
128	38	Chronic alcoholic psychosis; two months' duration..	3/ 3/27	7	14
129	37	Acute hallucinosis	3/28/27	6	11
130	59	Chronic alcoholic psychosis; marked mental deterioration; generalized arteriosclerosis.....	1/20/27	6	14
131	39	Chronic alcoholic psychosis; marked scoliosis; chronic fibroid phthisis	1/10/27	7	18
132	55	Alcoholic psychosis—delirium tremens; temperature, 99.8; pulse, 82; respiration, 24; marked toxicity....	1/11/27	10	24
		Patient mentally clear	1/30/27	8	16
133	52	Alcoholic psychosis—delirium tremens; temperature, 100; pulse, 84; respiration, 26.....	3/ 4/27	14	23
		Patient's mental picture clear.....	4/ 2/27	6	14
134	39	Alcoholic psychosis—delirium tremens; patient markedly toxic; unable to walk; temperature, 99; pulse, 84; respiration, 20; some optic atrophy.....	1/13/27	13	26
		Mentally clear; gait somewhat improved.....	2/21/27	8	16
135	45	Chronic alcoholic psychosis; bilateral pulmonary tuberculosis with cavitations; temperature, 101; pulse, 86; respiration, 26.....	3/ 7/27	13	31

the patient. In cases 110, 111, 115, 116 and 117 the test was done as a routine measure. As far as is known, these patients did not present complaints. It was the marked acceleration of the sedimentation reaction that led to further examination.

In not one case of dementia praecox was there a markedly increased reading unless the mental condition was complicated by some definite somatic disease.

The highest readings occurred in the acute types (delirium tremens and hallucinosis) of alcoholic psychoses, as in cases 129, 132, 133 and 134 (table 4). Cases 130, 131 and 135 are complicated by other physical conditions. It is interesting to observe in cases 132, 133 and 134 how the acceleration of the sedimentation reaction decreased with the mental improvement. The second readings, nevertheless, were still increased,

perhaps due to the remaining generalized toxicity and possible destruction of cells in the body. The cases of chronic alcoholic psychoses, unless accompanied by marked mental deterioration, tended to give normal readings.

From tables 5 and 6, it will be seen that in senile psychoses and in the psychoses due to cerebral arteriosclerosis the readings were increased.

TABLE 5.—*Readings in Senile Psychoses*

No.	Age	Sex	Clinical Diagnosis and Condition	Date	Sedimentation, Per Cent 1 Hr.	Sedimentation, Per Cent 2 Hrs.
136	72	♂	Simple deterioration; poor physical condition..	3/14/27	3	9
137	77	♂	Simple deterioration; general arteriosclerosis....	3/ 2/27	4	10
138	68	♂	Simple deterioration; physical condition fair....	3/ 2/27	5	9
139	72	♂	Simple deterioration; general arteriosclerosis....	3/ 2/27	5	12
140	74	♂	Paranoid type; general arteriosclerosis.....	1/11/27	5	14
			Patient failing physically and mentally.....	3/14/27	6	17
141	68	♂	Simple deterioration; fractured right clavicle...	3/ 3/27	9	24
142	73	♀	Simple deterioration; physical condition fair....	3/ 2/27	5	14
143	75	♀	Simple deterioration; general arteriosclerosis....	9/ 2/26	4	15
144	65	♀	Simple deterioration; physical condition fair...	3/14/27	7	16
145	85	♀	Simple deterioration; Colles' fracture in August, 1926.....	9/ 8/26	8	18
146	68	♀	Simple deterioration; physical condition fair....	10/13/26	8	18
147	68	♀	Paranoid type; general arteriosclerosis; cardiorenal disease	3/ 1/27	7	22
148	60	♀	Depressed and agitated type; cardiorenal disease	3/ 2/27	13	28
149	65	♀	Paranoid type; general arteriosclerosis; chronic otitis media	3/14/27	14	30
150	71	♀	Simple deterioration; general arteriosclerosis; cardiorenal disease.....	10/29-26	23	27

TABLE 6.—*Readings in Psychoses Due to Cerebral Arteriosclerosis*

No.	Age	Sex	Clinical Condition	Date	Sedimentation, Per Cent 1 Hr.	Sedimentation, Per Cent 2 Hrs.
151	66	♂	Minor cerebral insults; physical condition poor.	1/27/27	8	14
152	48	♂	Minor cerebral insults; chronic arteriosclerosis; cardiovalvular disease; chronic nephritis.....	1/13/27	4	9
			Patient had another cerebral insult; physical condition poor; temperature, 101; pulse, 98; respiration, 28.....	2/14/27	18	29
153	68	♂	Left hemiplegia; physical condition poor.....	1/13/27	5	13
154	45	♂	Left hemiplegia; chronic bronchitis, temperature, 99; pulse, 110; respiration, 24.....	4/ 4/27	6	15
155	62	♂	Minor cerebral insults; hypertension; emphysema; cardiorenal disease.....	3/14/27	8	15
156	58	♀	Right hemiplegia; hypertension; cardiorenal disease; pulse, 84.....	3/ 2/27	9	19
157	82	♀	Multiple cerebral insults with paraplegia; cardiorenal disease; temperature, 98; pulse, 88; respiration, 26.....	3/ 2/27	10	21

All general paralytic patients gave increased readings (table 7). Of the patients inoculated with malaria, patient 165 was the only one who died. Incidentally, this sedimentation reaction was the most accelerated of the group. Whether or not a markedly increased reading is a contraindication to inoculation of malaria cannot be stated at present. This question is worthy of further attention.

In fourteen cases of manic-depressive psychoses listed in table 8, all readings were within normal limits.

TABLE 7.—*Data in General Paralysis*

No.	Age	Sex	Clinical Condition	Date	Sedimentation, Per Cent 1 Hr.	Sedimentation, Per Cent 2 Hrs.
158	52	♂	Physical condition fair; blood and spinal fluid Wassermann reactions positive; typical general paralytic curve	1/20/27	4	9
159	49	♂	As in case 158	1/30/27	4	9
160	46	♂	Aortic regurgitation; blood and spinal fluid gave positive reactions; general paralytic curve	11/15/27	4	11
161	46	♂	Aortic regurgitation and stenosis; blood and spinal fluid reactions positive; paralytic curve.	1/20/27	5	12
162	32	♂	Marked grandiose ideas; blood and spinal fluid reactions positive; paralytic curve	1/14/27	4	13
163	43	♂	Marked mental deterioration; typical paralytic symptoms	3/14/27	6	16
164	45	♂	Marked mental deterioration; positive serologic and neurologic symptoms	1/17/27	8	14
165	61	♂	Aortic regurgitation; right inguinal hernia; arteriosclerosis; scoliosis and typical general paralytic symptoms. Patient was inoculated with malaria the same day; he died after the third chill.	11/15/26	30	47
166	29	♀	Blood and spinal fluid reactions positive; paralytic curve present; physical condition fair	10/ 3/26	6	13
167	55	♀	Serologic reactions positive; fracture of left hip in 1913	10/13/26	7	17
168	43	♀	Neurologic and serologic symptoms consistent with diagnosis	3/ 2/27	8	18
169	48	♀	Physical condition poor; marked spinal as well as cerebral involvement	9/ 8/26	17	33
170	35	♀	Mental, neurologic and serologic symptoms consistent with general paralysis; patient had perforated septum; gumma of right radius	3/ 7/27	23	41.5

TABLE 8.—*Readings in Manic-Depressive Psychoses*

No.	Age	Sex	Clinical Diagnosis and Condition	Date	Sedimentation, Per Cent 1 Hr.	Sedimentation, Per Cent 2 Hrs.
171	37	♂	Depressed type; physical condition good	1/29/27	1	3
172	40	♂	Depressed type; physical condition good	3/ 3/27	1	4
173	37	♂	Manic type; physical condition good	1/23/27	2	4
174	42	♂	Mixed type; fourth attack	3/17/27	1	4
175	28	♂	Manic type; second attack	3/21/27	3	5
176	26	♂	Manic type; second attack; patient disturbed. Patient fully recovered	2/ 5/27 3/ 4/27	3 2	7 5
177	27	♀	Depressed type; no complications	9/ 2/26	2	4
178	40	♀	Depressed type; no complications	9/ 2/26	3	5
179	50	♀	Depressed type; several previous attacks	10/13/26	2	7
180	61	♀	Mixed type; several previous attacks	8/19/26	4	7
181	62	♀	Depressed type; no complications	9/ 2/26	3	7
182	30	♀	Mixed type; no complications	9/ 8/26	4	9
183	46	♀	Mixed type; several previous attacks	9/ 8/26	4	11
184	67	♀	Depressed type; six previous attacks; general arteriosclerosis	9/ 8/26	5	12

TABLE 9.—*Readings in Cases of Involutional Melancholia*

No.	Age	Sex	Clinical Diagnosis and Condition	Date	Sedimentation, Per Cent 1 Hr.	Sedimentation, Per Cent 2 Hrs.
185	62	♂	Physical condition good	1/13/27	3	5
186	46	♂	Physical condition good	1/13/27	3	6
187	46	♂	No complications	1/13/27	2	6
188	54	♀	No complications	9/ 2/26	2	4
189	47	♀	No complications	10/ 1/26	1	7
190	45	♀	No complications	10/ 1/26	1	7
191	55	♀	Physical condition good	9/20/26	4	10
192	53	♀	No complications	8/18/26	5	13
193	52	♀	Marked mental deterioration	10/13/26	6	18
194	60	♀	Marked mental deterioration	9/ 3/23	6	19
195	43	♀	Marked mental deterioration	3/14/27	9	19
196	45	♀	Question of endocrine disorder	4/ 1/27	7	20
197	47	♀	Patient presented no complaints, but on 3/30/27 the ear began to discharge; question of tuberculous origin	3/ 7/27	9	20

In thirteen cases of involutional melancholia recorded in table 9, the readings vary. In a few cases the reactions were normal, but the tendency was toward a slight increase in the readings. The cases in which the readings were most increased were accompanied by marked mental deterioration or by definite physical disorders.

TABLE 10.—*Readings in Other Types of Psychoses*

No.	Age	Sex	Clinical Diagnosis and Condition	Date	Sedimentation, Per Cent	
					1 Hr.	2 Hrs.
198	47	♂	Paranoia	1/23/27	2	4
199	33	♂	Paranoia	1/17/27	2	5
200	38	♀	Paranoia	2/ 3/27	3	8
201	45	♀	Paranoia; radical amputation of the breast in 1923	8/27/26	3	9
202	37	♂	Psychoneurosis—hysterical type	1/ 3/27	2	4
203	30	♂	Psychoneurosis—psychasthenic type	3/30/27	3	5
204	24	♀	Psychoneurosis	11/ 5/26	3	7
205	45	♀	Psychoneurosis—psychasthenic type	9/20/26	4	10
206	20	♀	Psychopathic personality	9/19/26	2	4
207	35	♂	Epileptic psychosis	1/13/27	1	2
208	53	♂	Epileptic psychosis; marked mental deterioration	1/13/27	3	9
209	51	♂	Epileptic psychosis; marked mental deterioration	1/13/27	3	11
210	40	♀	Epileptic psychosis; no complications	9/13/26	3	7
211	39	♀	Psychosis due to drugs; patient clear mentally.	9/ 8/26	4	9
212	40	♀	Psychosis due to drugs; patient toxic; temperature, 99.8; pulse, 84	9/ 8/26	22	34
			Patient's mental picture almost clear; pulse, 82.	9/30/26	12	22
213	58	♂	Psychosis with other disease of the brain—cerebral embolism; systolic blood pressure 160; diastolic, 90	1/29/27	3	8
214	18	♂	Psychosis with encephalitis lethargica	1/13/27	3	8
215	52	♂	Psychosis with amyotrophic lateral sclerosis	3/ 3/27	15	28
216	46	♂	Traumatic psychosis; fractured skull, October, 1926; patient mentally clear	3/21/27	1	4
217	38	♂	Traumatic psychosis; injury about one year before; blurring of nasal borders of disks	3/ 3/27	5	12
218	66	♀	Psychosis with somatic disease—obscure anemia; red blood cells, 3,000,000; hemoglobin, 65; pulse, 110	2/18/27	15	34
219	61	♀	Psychosis with cardiorenal disease; hypertension; arteriosclerotic cardiovalvular disease with chronic nephritis	8/29/23	20	32
220	63	♀	Psychosis with diabetes; blood chemistry and urinary symptoms consistent with diabetes mellitus	3/14/27	33	50
221	22	♂	Psychosis with mental deficiency	3/ 3/27	4	10
222	37	♂	Psychosis with mental deficiency	1/15/27	7	14
223	20	♂	Psychosis with mental deficiency	1/20/27	4	12
224	48	♂	Psychosis with mental deficiency	1/23/27	8	15
225	42	♀	Psychosis with mental deficiency	9/ 2/26	4	13
226	61	♀	Psychosis with mental deficiency	8/18/26	7	17
227	38	♀	Psychosis with mental deficiency; gonorrhea	9/30/26	11	26
228	23	♀	Psychosis with mental deficiency; internal hydrocephalus	11/ 5/23	10	23

In table 10 are given the results in thirty-two cases of psychoses of types other than those hitherto mentioned. In all cases of psychopathic personality, psychoneuroses and paranoia the readings are within normal limits. On the other hand, there is acceleration of the sedimentation reaction in all cases of psychoses due to organic disease of the brain or nerves, psychoses due to somatic disease and psychoses showing mental deficiency. With the exception of one, all cases of epileptic psychoses showed increased readings.

COMMENT

Because the erythrocyte sedimentation reaction is a reliable and efficient detector of many somatic disorders, it is of practical value in psychiatric work. In dealing with the insane, especially at state hospitals, one is apt to overlook physical disorders. This is due chiefly to the lack of subjective complaints on the part of the patient because of the mental condition. In cases 110, 111, 115, 116 and 117, the somatic diseases were accidentally discovered by the sedimentation test. These patients did not complain of discomfort.

CONCLUSIONS

1. The acceleration of the erythrocyte sedimentation reaction varies directly with the amount of mental deterioration, the amount of organic destruction and the amount of toxicity present.

2. Unless the cases are complicated by physical disease, the sedimentation reactions are normal in cases of manic-depressive psychoses, psychopathic personalities, psychoneuroses and paranoia.

3. Increased readings are found in all cases of senile psychoses, psychoses due to cerebral arteriosclerosis, general paralysis, neurosyphilis, psychoses showing mental deficiency or somatic disease, acute types of alcoholic psychoses and in many cases of epileptic psychoses and involutional melancholia.

4. In dementia praecox, the simple and paranoid types tend to give normal readings, while the hebephrenic and chiefly the catatonic types tend to give slightly accelerated reactions. Cases showing marked mental deterioration give increased readings. No case of dementia praecox gave a marked increase unless complicated by some physical disorder.

5. The sedimentation reaction is of limited aid in psychiatric diagnosis.

6. Repeated tests are helpful in determining the clinical condition of the patient.

7. Because the sedimentation test is a reliable and efficient indicator of many somatic diseases, it is of practical value in dealing with psychotic patients.

LONG SECTION BLOOD SUGAR TOLERANCE STUDY IN A CASE OF DEPRESSION *

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In recent years, a number of studies [1] have been published indicating the occurrence of a certain apparently rather typical disturbance of carbohydrate metabolism in depressions of the manic-depressive type. In several instances, it has been suggested that such a disturbance [2] is seemingly phasic or concomitant with the mental difficulty, and disappears when the patient recovers from the latter. This question is of significance, as it may have important bearing on the concept or theory of the psychosis and suggests an important implication of the so-called physiologic level, in relation to what might be casually dismissed as simply a mental condition.

In view of this, we felt that it might prove of some interest to report a complete study of such a case from this standpoint in the hope that it might be of some assistance in establishing in fact the suggestion already made in the literature. The case itself, as will be seen, is paradigmatic in all respects: genetically, somatically and psychiatrically. The chemical observations were carried out under carefully controlled conditions over a long period of time and through all phases of the disorder; this was distinctly an advance over previous work, in which hardly enough observations were reported to establish a true long section status. It is in view of these factors particularly, the unique typicality of the case and the completeness of the study in the light of the admitted importance of the problem that this report is made.

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* From the State Psychopathic Hospital.

1. Raphael, T., and Parsons, J. P.: Blood Sugar Studies in Dementia Praecox and Manic-Depressive Insanity, Arch. Neurol. & Psychiat. **5**:687 (June) 1922. Henry, G. W., and Mangam, E.: Blood in Personality Disorders, ibid. **13**:743 (June) 1925. Olmstead, W. H., and Gay, L. P.: Study of Blood Sugar Curves Following a Standardized Glucose Meal, Arch. Int. Med. **29**:384 (March) 1922. Drury, K. K., and Farran-Ridge, C.: Some Observations on the Types of Blood Sugar Curve Found in Different Forms of Insanity, J. Ment. Sc. **71**:8 (Jan.) 1925. Mann, S. A.: Blood Sugar Studies in Mental Disorders, J. Ment. Sc. **71**:443 (July) 1925.

2. Drury, K. K. (footnote 1). Mann, S. A. (footnote 1).

REPORT OF CASE

Clinical History.—W. J. B., aged 56, was admitted to the State Psychopathic Hospital on April 1, 1925, because of depressed reactions which developed subsequent to the death of his wife. Analysis of the family history revealed, on the paternal side, numerous persons of cycloid make-up with frequent instances of frankly manic-depressive disorder determinable through many generations. One of the patient's daughters had passed through two distinct attacks of manic-depressive excitement. Between the ages of 23 and 48, the patient had suffered six episodes of mental disorder, which were typically manic-depressive. During the last of these episodes, he was treated at the State Psychopathic Hospital over a period of eight months. His condition at that time was classically one of depression, which was well marked in mood and in thought content, and which was accompanied by psychomotor retardation.

Examination.—Physical examination on the occasion of admission for the seventh or present attack gave essentially negative results with reference to clinically determinable somatic disease. From the standpoint of somatic constitution or bodily habitus, however, the patient was strikingly of the so termed pyknic or macrosplanchnic type thought to be characteristic[3] of manic-depressive patients. The morphologic index was 252, chest circumference — stature index, 581; the bi-iliac-stature index, 182; the thoracic index, 566, and the subcostal angle, 97 degrees; all figures indicative of large trunk dimensions in relation to linear or limb values, which is the effective criterion for this habitus.

Neurologic and routine laboratory examinations gave negative results.

Psychiatrically, the picture presented was typically one of uncomplicated manic-depressive depression with especially pronounced psychomotor retardation. During the first period of residence in the hospital, the disorder was rather mild. After a time, the depression became profound; however, it gradually abated, until about seven months after admission, when the patient was discharged much improved. He returned to the hospital about a month later for follow-up examination; at this time, he was thought to have almost recovered.

Blood Sugar Studies.—During the course of the patient's disorder, carbohydrate metabolism was carefully studied. For this purpose the tolerance for sugar was tested according to the procedure of Janney and Isaacson.[4] The actual determinations of the blood sugar were made in accordance with the technic of Folin and Wu.[5] In all, fifteen tolerance analyses were made; a number of determinations of the basal metabolism were also made. A careful record was kept of the clinical observations, and all observations were conducted under uniform and, as far as possible, basal conditions.

RESULTS

The determinations are presented in tabular form. From the table and accompanying chart, it is apparent that during the beginning phase of the disorder, before the depression had become especially profound,

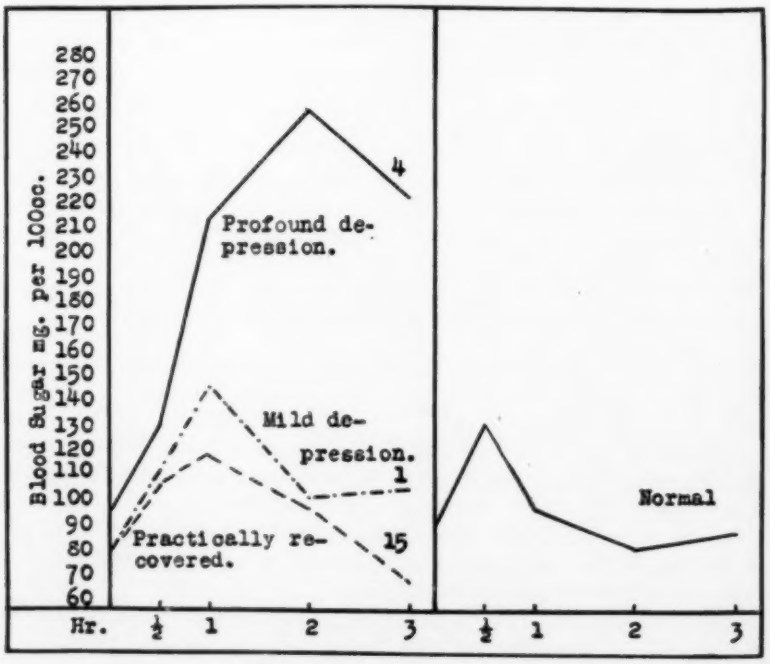
3. Kretschmer, E.: Körperbau u. Charakter, Berling, Julius Springer, 1921.

4. Janney, N. W., and Isaacson, V. I.: A Blood Sugar Tolerance Test, J. A. M. A. **70**:1131 (April 20) 1918.

5. Folin, O., and Wu, H.: A System of Blood Analysis: Supplement I. A Simplified and Improved Method for Determination of Sugar, J. Biol. Chem. **41**:367 (March) 1920.

TABLE 1.—*Glucose Tolerance Curves After the Ingestion of 1.75 Gm. of Glucose Per Kilogram of Body Weight*

No.	Date, 1925	Phase of Beginning Depression	Weight, Lb.	Blood Sugar: Fasting, Mg.	Blood Sugar: After Ingestion of Glucose: ½ Hr. Mg.	1 Hr. Mg.	2 Hr. Mg.	3 Hr. Mg.	Basal Metabolic Rate, per Cent
1	4/ 4	Very mild depression	217	82	114	148	102	106	
	5/11	Mild depression	...	...	...	...	...	...	— 8.5
2	5/13	Depression slightly greater	207	94	163	172	118	114	
		Average		88	139	160	110	110	
		Phase of Profound Depression							
3	6/ 3	Depression marked	202	102	135	209	157	137	
4	6/10	Depression profound	193	98	132	216	258	224	
5	6/17	Depression slightly less profound	181	100	127	188	180	171	
		Average		100	131	204	200	177	
		Phase of Recovery							
	6/22	Improved	...	...	...	...	...	...	— 7.4
6	6/24	Less depressed	180	80	108	143	147	143	
7	7/ 1	No change	178	75	103	108	97	88	
	7/ 3	No change	...	...	...	...	...	...	— 5.0
8	7/ 8	Condition improved	180	73	75	102	90	69	
	7/10	Condition improved	...	...	...	...	...	...	—11.7
9	7/15	Not much change	184	69	75	91	77	73	
10	7/22	Improved	186	71	85	103	79	76	
11	7/29	Little change	184	70	89	101	76	68	
12	8/ 5	Improved	184	69	77	85	76	74	
13	9/12	Improved	186	76	89	73	64	80	
	10/19	Improved	...	...	...	...	...	...	—11.0
14	10/28	Continues to improve	190	68	73	91	67	52	
15	12/22	Practically recovered	192	82	109	120	98	68	
		Average		73	87	101	87	79	



Glucose tolerance curves.

the blood sugar tolerance was definitely although relatively slightly deviant from the average or normal [6] formula. This deviation showed itself essentially in a higher blood sugar peak following the ingestion of glucose and a delay in return to the fasting level, normally apparent in one and one-half or two hours.

During the period of profound depression, these features were much accentuated, and there was also some indication of elevation of the fasting level. This general type of response in depressed states has been observed previously.[1]

In the third phase, that of recovery, a further change was seen: the peak of the curve for tolerance of sugar was much lower, and there was more prompt return to the fasting level with a tendency toward a definite lowering of the latter; that is, there was an approximate return to the normal response, except, perhaps, that the fasting level continued a trifle low. The last curve of the series (15), secured a month after the patient was discharged and when he attained his highest point of clinical improvement, was especially like the average in pattern, although a slight tendency toward retardation was still evident. It is conceivable that had the patient recovered completely, this feature might have been absent. It might be mentioned also that several "flat" curves were obtained during the period of improvement; the exact significance of these is not apparent. Of interest in this connection, however, is the fact that similar curves [1] have been obtained in manic states; this suggests the possibility of such a physiologic cycle in connection with clinical recovery, without apparent evidence, however, of frankly manic psychiatric reactions.

The basal metabolic rate, as might have been expected,[7] was slightly diminished throughout the period of study, although it did not show any essential variation in relation to fluctuation in the clinical phase.

COMMENT

A case of manic-depressive depression, typical from all points of view, has been studied from the standpoint of blood sugar tolerance. The observations bear out and lend added weight to previous observations on this psychosis. In this disorder there is a tendency toward an

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6. Foster, G. L.: I. Some Comparisons of Blood Sugar Concentrations in Venous Blood and in Finger Blood, J. Biol. Chem. **55**:291 (Feb.) 1923. II. An Interpretation of the Blood Sugar Phenomena Following the Ingestion of Sugar, ibid., p. 303.

7. Gibbs, C. E., and Lemcke, D.: Basal Metabolism in Dementia Praecox and Manic-Depressive Psychoses, Arch. Int. Med. **31**:102 (Jan.) 1923. Bowman, K. M., and Grabfield, G. P.: Basal Metabolism in Mental Disease, Arch. Neurol. & Psychiat. **9**:358 (March) 1923. Farr, C. B.: Results of Basal Metabolism Tests in One Hundred Mental Cases, ibid. **12**:518 (Nov.) 1924.

abnormal reaction following the ingestion of glucose; this is shown by a curve of decreased tolerance resembling that found in certain somatic disturbances, such as diabetes mellitus. This abnormality of carbohydrate metabolism appears to be phasic and to disappear with clinical recovery. It therefore seems that in this condition the patient is affected physiologically as well as psychologically and this paper, if it does nothing else, emphasizes the concept of the "total" nature or quality of conduct.

Whether this physiologic factor is primary or secondary to the mental symptomatology is problematic, but there can be no question that the association is close. It is of some interest too also in this connection that not infrequently this type of disorder or shifting in its phase seems to occur without apparent psychogenic motivation or a significant objective cause. In our case the psychic factor may well have been primary, since the psychosis seems to have been precipitated by a definitely objective and affect-disturbing occurrence, the death of the patient's wife. At any rate judging by the course followed by the patient, it seems impossible to separate one factor from the other, so closely do the two seem to have been parallel.

As to the actual mechanism concerned in the pathophysiology encountered, little can definitely be said. In view of the diminished basal metabolic rate, a general metabolic depression may be postulated, of which the disturbance in carbohydrate physiology represents merely a single manifestation. It appears probable that the vegetative and endocrine systems may be implicated, possibly in part through central influence;[8] that hepatic function may also be involved in some way is not inconceivable. Interesting problems, likewise, are the possible rôles of the psychomotor retardation and the affective element of the psychosis as such in relation to the physiologic disturbance noted. A pertinent question in this connection is the possible significance of the patient's constitution in view of its more or less recognized morphologic importance in this disorder.

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8. Raphael, T.; Searle, O. M., and Ferguson, W. G.: The Amyostatic Syndrome of Epidemic Encephalitis, Arch. Neurol. & Psychiat. **15**:103 (Jan.) 1926. Gurdjian, E. S.: On Certain Functions of the Hypothalamus, to be published.

THE KAFKA PARAFFIN REACTION IN THE SPINAL FLUID*

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A never ending succession of various modifications of the original colloidal test as developed by Lange presents itself to those who are interested in the pathologic phenomena of the spinal fluid. Solomon states that, useful as they may be, none of these colloidal reactions add anything new to our knowledge of the spinal fluid. All of these tests have their supporters, whose bias in some instances is suspiciously tinctured by nationalistic feeling, particularly in the case of the benzoin test as devised by Guillain and Lechelle.

To the clinician the complex interplay of colloidal substances still remains a matter of empiric observation rather than of understanding. A number of writers have attempted to interpret the various curves from the biochemical point of view but few have ventured sufficiently into the realm of physical chemistry to account for the nonconcomitance of the various protein tests, which is frequently found and which has been particularly emphasized by Solomon [1] and Fremont-Smith.[2]

Such factors as changes in hydrogen ion concentration, variations in the iso-electric points, differences in the proportions of suspensoids and emulsoids have been investigated as possible causes of differential precipitations. While the exact significance of each may not be readily understood, any or all of these protein tests may demonstrate certain minute deviations from the normal. One need only mention Neel's [3] interpretation of the Pandy and Nonne-Apelt tests; Weichbrodt's [4] conception of the sublimate reaction in syphilitic fluids, Levinson's [5] differential precipitation in the meningitidites, and Boltz's [6] acetic-anhydride

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1. Solomon, H. C.: Nonconcomitance of Spinal Fluid Tests, Arch. Neurol. & Psychiat. **3**:49 (Jan.) 1920.

2. Fremont-Smith, F.: The Human Cerebrospinal Fluid, New York, Hoeber, 1926, p. 100.

3. Neel, A. V.: Schweiz. Arch. f. Neurol. u. Psychiat. **17**:3, 1925.

4. Marx, E. J.: Weichbrodt's Sublimat Reaction in Cerebrospinal Fluid, Monatschr. f. Psychiat. u. Neurol. **48**:227, 1920.

5. Tashiro, S., and Levinson, A.: J. Infect. Dis. **21**:571, 1917.

6. Boltz, O. H.: Studies of the Cerebrospinal Fluid with Acetic-Anhydride Sulphuric Acid Test, Am. J. Psychiat. **3**:1, 1923.

sulphuric acid test in fluids of general paralytics. To these may be added the quantitative test of Denis and Ayer, and finally, the various colloidal tests.

In these days of ultraspecialization, it has been repeatedly emphasized that the diagnosis is too often a synthesis of the disconnected opinions of many minds. Ayer [7] stresses the necessity, for example, of one person carrying out all the important procedures in a given case in order that a better interpretation may be made, and illustrates the possible errors arising when the spinal fluid passes through too many hands. In the average hospital routine, it is found advisable to urge the intern to carry out all procedures and analyses himself in order to arrive at a more rounded-out clinical opinion.

In our experience, it has been found that the most practicable colloidal test is the gum mastic reaction. Acccording to Keidel and Moore,[8] Wassermann [9] and others, this test is valuable as an integral part of the study of the spinal fluid, largely because of the simplicity of the preparation of the reagents and their stability in the face of minor technical errors; it therefore is admirably suited for the small laboratory attached to the ward. The method used in our series is that of Stanton, and the numeral nomenclature that of Keidel and Moore.

In 1925, Kafka [10] suggested the use of paraffin as a colloidal reagent and claimed that it was as valuable as the Lange preparation and had all the advantages of the gum mastic reaction. Its peculiar advantages were its purity, cheapness and simplicity of chemical structure. The technic follows.

TECHNIC

Dissolve 0.3 Gm. of American paraffin, with a melting point of from 50 to 52 C. in 100 cc. of absolute alcohol, by heating. On cooling, some of the paraffin will be deposited. This mother solution is mixed quickly with an equal amount of distilled water at a temperature exactly that of the melting point of the paraffin. This results in a stable, turbid fluid appearing not unlike the mastic emulsion. Ten tubes are used. In the first is placed 0.75 cc. of 0.3 per cent sodium chloride solution, and 0.5 cc. in each of the other nine tubes. To the first tube is added 0.25 cc. of the spinal fluid; 0.5 cc. is then pipetted from the first to the second tube and then the same amount through the others as in any other colloidal test. Finally, 1 cc. of the paraffin solution is added to each tube. The tubes are allowed to stand over night at room temperature.

We have modified the reading of the Kafka curve to make simple a comparison with the gum mastic and recognized five instead of six

7. Ayer, J. B.: The Analysis of Spinal Fluid Tests, J. A. M. A. **87**:6 (Aug. 7) 1926.

8. Keidel, A., and Moore, J. C.: Comparative Results of Colloidal Mastic and Gold, Arch. Neurol. & Psychiat. **6**:163 (Aug.) 1921.

9. Wassermann, H.: Comparative Results of Colloidal Gold and Colloidal Mastic Tests, Arch. Int. Med. **33**:401 (March 15) 1924.

10. Kafka, V.: Ztschr. f. d. ges. Neurol. u. Psychiat. **94**:652, 1925.

degrees of precipitation. In a general paralytic curve reaction, the emulsion clears and the paraffin settles at the top. This feature offers a unique advantage over the gum mastic reaction, particularly in the study of fluids with a high albumin content. In such a case the albumins settle to the bottom, and are readily differentiated from the indicator substance. In paralytic fluids with relatively small amounts of albumin, the paraffin solution frequently resisted precipitation in the first tube, owing to this protein fraction. Such was not the case in the corresponding mastic curve.

In this series, ninety-one fluids were examined and parallel readings were made of the gum mastic and the Kafka paraffin reactions. The results of the more familiar protein tests, being too well known to be of interest, need no comment, but the Boltz reaction known as the acetic-anhydride sulphuric acid which has, so far, not been regarded as a routine test, was performed in every instance along with the colloidal tests and is worthy of note. According to its originator, it reacts to the same fraction as is detected by the Noguchi test and he further claims for it a specificity for syphilitic fluids. More recently, Grossman [11] and Harris [12] confirmed his observations in that they found it positive in almost every case (97 per cent) of general paralysis.

RESULTS

In General Paralysis.—Thirty-five specimens of fluid were examined and the results showed five dissimilar readings, but none of these were sufficiently different to be noteworthy. Eight of the Kafka reactions demonstrated an incomplete precipitation in the first tube, while such a phenomenon did not occur in the corresponding gum mastic test. The explanation offered is that the Kafka reagent is more easily protected by emulsoids which occur in greatest amount in the first tube. In all cases showing atypical curves, the patients were under treatment with tertian malaria. Twenty-one, or 60 per cent, of all these fluids showed a positive acetic-anhydride and sulphuric acid test, a figure considerably lower than the 100 per cent found by Boltz.

In Tabes and Taboparalysis.—In this series, ten specimens of fluid were studied. The same diversity of results was obtained here as was found by Cockrill [13] in the comparison between the gold, mastic and benzoin tests. Two fluids showed dissimilar reactions, and the results again confirm the opinion that there is no typical or constant tabetic curve. The acetic-anhydride sulphuric acid test was positive in 6 or 60 per cent of the cases.

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11. Grossman, S.: J. Ment. Sc. (July) 1925.
12. Harris, J. S.: Brit. M. J. (Jan. 23) 1926.
13. Cockrill, J. R.: Comparison of Gold Benzoin and Mastic Tests on Spinal Fluid, Arch. Neurol. & Psychiat. **14**:455 (Oct.) 1925.

In Syphilis of the Central Nervous System.—Four fluids, giving positive reactions with the Wassermann test, showed three positive acetic-anhydride sulphuric acid tests, and the colloidal readings were fairly parallel.

In Meningitis.—In this series, eleven fluids, including tuberculosis, pneumococcus and streptococcus meningitis were compared. The meningeal "peak" was more pronounced in the gum mastic test in all fluids except one. One fluid failed to demonstrate a typical curve with the Kafka test. In all but two cases, the acetic-anhydride sulphuric acid test was strongly positive and more pronounced than in the fluids of syphilitic patients. Our experience with the Boltz test emphasizes its value in such cases as early tuberculosis and septic meningitis.

In Extraneural Syphilis.—Nine specimens of fluid were found with a remarkable similarity in the mastic and Kafka readings. Only two were entirely negative. The acetic-anhydride sulphuric acid test was consistently negative.

In Neurologic Conditions.—Various neurologic disturbances, such as tumor of the brain, tumor of the spinal cord with a Froin syndrome, multiple sclerosis and epilepsy demonstrated the greatest diversity of results. Two cases of multiple sclerosis are characterized by non-paralytic readings. Various protein tests on these fluids emphasized the perplexing nonconcomitance of these reactions. The acetic-anhydride sulphuric acid test was positive in the case of brain and of cord tumor.

In the Psychoses.—Fifteen specimens of fluid from various patients suffering from psychoses, a few of which may have had some organic basis, were studied. In none of these was the acetic-anhydride sulphuric acid test positive; thirteen, however, showed a positive phase 2 of Nonne-Apelt reaction, and about the same percentage showed a slightly positive colloidal curve with both the mastic and the Kafka tests. The parallelism in this group was most pronounced. It emphasized the danger incident to the interpretation of the so-called "syphilitic zone" curve.

CONCLUSION

The Kafka paraffin colloidal test on spinal fluid is recommended as a simple and reliable procedure, which is admirably suited to the small laboratory attached to the hospital ward and to the private office. The test compares favorably with that of the gum mastic as far as results are concerned. The technic, however, is a little more difficult because of the temperature requirements.

TOXIC INFECTIOUS DISEASE IN THE PERIPHERAL AND CENTRAL NERVOUS SYSTEM— SO-CALLED NEURONITIS

A CLINICAL STUDY WITH SUGGESTIONS AS TO NOMENCLATURE *

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Our object in this paper is two-fold: first, to present a group of cases in which toxic or infective processes affect both the peripheral nerves and the nerve cells and tracts of the spinal cord; second, to discuss the nomenclature of these conditions.

For a number of years, it has been known that the lesions in some cases of so-called "peripheral neuritis" have involved the central as well as the peripheral nervous system. In 1898, Mills [1] realized this and endeavored to express the idea in terms of the neuron theory then being brought forward; he coined the term "neuronitis," noting that it had an "unnatural" sound. Since that time, numerous writers have emphasized this association of pathologic changes in the peripheral nerve and in the central system. In this country, Kraus [2] stressed the concurrence of these lesions in diabetes mellitus; Hyslop and Kraus,[3] in palsies caused by lead poisoning and Foster Kennedy,[4] in certain types of epidemic encephalitis which he called "infective neuronitis." Abroad, Walshe [5] postulated involvement of the anterior horn cells in diphtheritic neuritis, believing that the toxin is carried along the perineural lymph channels of the peripheral nerves from the site of the local lesion—a lymphogenous "toxi-infection of the nervous system" (Orr and Rows).

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* From the Neurological Service of Bellevue Hospital, Dr. Foster Kennedy, Director.

1. Mills, C. K.: The Reclassification of Some Organic Nervous Diseases on the Basis of the Neuron, J. A. M. A. **31**:11 (July 2) 1898.

2. Kraus, W. M.: Involvement of the Peripheral Neurons in Diabetes Mellitus, Arch. Neurol. & Psychiat. **7**:202 (Feb.) 1922.

3. Hyslop, G. H., and Kraus, W. M.: The Pathology of Motor Paralysis by Lead, Arch. Neurol. & Psychiat. **10**:444 (Oct.) 1923.

4. Kennedy, Foster: Infective Neuronitis, Arch. Neurol. & Psychiat. **2**:621 (Dec.) 1919.

5. Walshe, F. M. K.: The Forms of Peripheral Neuritis Among Troops Serving with Egyptian Expeditionary Force, 1915-1919, Brain **43**:74, 1920.

Feiling,[6] reiterating the fact, stated that the "infection or intoxication falls, in many cases, at all events, on the central and peripheral nervous system."

Practically all authors believe with Buzzard [7] "that this group might well have a distinctive name if only a suitable one could be found." He demurs at the use of the term "neuronitis." Still, certain cases seem restricted to the peripheral nervous system throughout their course, and may be called peripheral neuritis. In other instances, it is evident that the term peripheral neuritis is inadequate, because it fails to include the idea of associated pathologic alteration of the cord. Mills' term "neuronitis," revived by Kennedy, has not been generally accepted. Kraus [8] has attempted to extend this nomenclature by introducing the terms: (1) "cellular peripheral neuronitis," for disease of the cell bodies; (2) "truncular peripheral neuronitis, radicular and neural types," for processes affecting the root and trunks of the nerves, and (3) "terminal peripheral neuronitis," for the more distal involvement of the nerve trunks.

The word "neuronitis" connotes inflammation of a neuron—i. e., of a nerve cell and its processes. It is generally admitted that inflammation is a reaction of the tissues involving the blood vessels and the connective tissue stroma as well as the parenchymal cells, and that inflammation restricted to a parenchymal cell does not exist; hence, the "unnatural sound" of the word. It is easy to criticize this terminology, but it is difficult to proffer a better one.

We suggest the following: (1) peripheral neuritis for those cases in which only the distal parts of the nerve trunks are affected; (2) radiculitis for involvement of the roots, and (3) central neuritis for involvement of the cells and tracts of the neuraxis, the part to be designated by the appropriate adjective—spinal, medullary, pontile, etc. Occasional cases will require a caption compounded of two of these terms.

In this use of the word, neuritis means inflammation of nerve tissue in the same sense that phlebitis signifies a similar reaction in the tissues making up a vein. Such usage is sanctioned by the present conceptions and teaching in pathologic anatomy.

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6. Feiling, A.: Multiple Neuritis, Oxford Loose Leaf System of Medicine **6**:647, 1921.

7. Buzzard, E. F.: On Certain Acute Infective or Toxic Conditions of the Nervous System, Brain **30**:85, 1907.

8. Kraus, W. M.: The Non-Traumatic Diseases of the Peripheral Nerves, Nelson Loose Leaf Living Medicine, 1926, vol. 6, p. 331.

The term central neuritis has been used before in a different connection. In 1901, Adolf Meyer[9] described a clinical and pathologic condition under this caption. He says:

> It is a partially systemic but widely distributed and strikingly symmetrical parenchymatous alteration of numerous nerve elements, chiefly of the cortico-thalamic connections of the motor area, the auditory radiation, the forceps, the pyramids, the fillet, the restiform body and, to a lesser degree, the posterior columns of the cord, the intersegmental elements, and the segmental efferent motor elements. The change is that of axonal reaction in many of the large cell types and decay of the myelin sheaths. Instead of the long descriptive term, "Partially systemic parenchymatous degeneration principally of the central nervous system," we propose the expression, "central neuritis," in the sense of an equivalent of parenchymatous neuritis, but mainly of central distribution.
>
> This alteration has been found to occur in peculiar forms or end-stages of depressive disorders, near or after the climacteric period, alcoholico-senile and alcoholico-phthisical cachectic states, idiocy, and perhaps also general paralysis. Ordinary infectious and cachectic states do not, however, appear to form an important link in the causes.
>
> Clinically, the symptoms are rather vague; after a course in which there is no suspicion of organic disorder, there appears, more or less suddenly, difficulty of locomotion, increasing weakness for coordinated movements, at times jactations of the limbs and rigidity and disorders of the reflexes, often together with diarrhea and occasional febrile fluctuations; the mental condition in this terminal episode is either that of anxious perplexed agitation, delirium, or stupor, similar to a protracted delirium tremens. The nature of the disorder appears to be an equivalent of the general type of parenchymatous nerve-cell alterations, but, in distinction from the well-known infectious and toxic types of "polyneuritis," of preeminently central distribution.

The symptoms of the syndrome described by Meyer were due to lesions in the brain and in the brain stem. Psychic disturbances dominated the clinical picture. The disease usually appeared in the fifth or sixth decade (the average age of his eight patients was 55 years) and always ended fatally after a few months or more. Meyer showed that the lesions were degenerative in nature.[10]

In our cases, the clinical evidence pointed to involvement of the peripheral nerves and the spinal cord. Neurologic signs were most prominent. The disease appeared earlier in life (the average age was 33 years), and no fatality occurred. The cases are not similar or even comparable in any way. Although we have not had any pathologic material, we believe that the clinical characteristics and course justify our conclusion that the pathologic substratum of our cases was essentially inflammatory.

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9. Meyer, A.: On the Parenchymatous Systemic Degeneration Mainly in the Central Nervous System, Brain **24**:47, 1901.

10. In a recent personal communication to the authors, Dr. Meyer writes: "I am quite sure that my central neuritis cases were food deficiency disorders, with the same histological findings as acute pellagra."

We would prefer to use the term "central neuritis" (with a qualifying adjective, viz., spinal, etc.) in our cases, because signs of central involvement, most often spinal, were added to those due to peripheral neuritis. We feel that the title "peripheral neuritis with central spinal neuritis" (as we have used it in some of the cases to be described) is especially apt because it draws attention to the inflammatory nature of the process, stresses the implication of two parts of the nervous system, and at the same time emphasizes the pathologic unity underlying the varied symptomatology.

Since the pathologic alterations in Meyer's cases were characterized by degenerative features, we should prefer to designate them by his own descriptive term, "partially systemic parenchymatous degeneration of the central nervous system."

The following illustrative cases indicate the way in which these terms have been applied.

REPORT OF CASES

CASE 1.—*A toxic infectious condition of the spinal cord associated with involvement of the peripheral nerves following a respiratory infection. Rapid recovery.*

History.—J. N., aged 37, a Hungarian housewife, entered Bellevue Hospital (neurologic service), March 28, 1926, complaining of inability to walk, general weakness, which was more marked in the lower extremities, a "cold, numb, wooden" sensation in the extremities, and urinary retention. The family history, habits and past history were irrelevant. The present illness began suddenly four weeks before admission to the hospital, with symptoms of an acute infection of the upper respiratory tract (marked malaise, cough, fever) which spread; the cough and malaise became aggravated. At the end of a week, the physician diagnosed pneumonia. In the third week, severe pains appeared in the back and all the extremities, and intense frontal and basal headaches developed. At this time the four extremities became weak and felt "numb, wooden and frozen." The same sensations were referred to the trunk to a less degree. In addition, urinary retention set in, making catheterization necessary. In the beginning of the fourth week there was considerable nausea and vomiting. The weakness affected the lower extremities more than the upper, and the right upper extremity much more than the left. No weakness was referred to the cranial innervations. In another week, improvement began in the motor functions of the extremities. Urinary retention persisted. At this juncture, the patient entered Bellevue Hospital.

Examination.—On April 3, 1926, the patient was bedridden and pale. The function of the cranial nerves was entirely normal. The upper extremities showed weakness on the right side to be greater than on the left, manifested especially in the grips. Atrophy or fibrillations were not noted. In the lower extremities, there was a marked bilateral weakness of the anterior tibial and hamstring groups of muscles; the quadriceps extensors were involved to a less degree. Atrophy was present, especially in the thighs; again, no fibrillations were seen. The reflexes of the upper extremities were equal and over-active. The ankle reflexes were absent; the knee reflexes were diminished, the left more than the right. The Babinski toe sign was not present. The abdominal reflexes were not elicited (the patient was a multipara).

Below the seventh dorsal dermatome there was general reduction of sensibility to touch, pain, temperature and vibration. The sense of joint position was intact. In addition, a still further reduction of these sensibilities was noted in the right upper and lower extremities, with a glove and stocking distribution. These changes were present, but were much less definite on the left side. Retention of urine still necessitated daily catheterization.

Course.—On April 6, the patient began to void and defecate voluntarily. On April 8, the knee reflexes became more active and the ankle reflexes barely reappeared. On April 12, control of the bladder was temporarily restored. On April 13, a definite Babinski toe reflex appeared on the right, and an equivocal one on the left. These abnormal reflexes persisted for several weeks. Within a few more weeks, normal function of the bladder was established. On April 15, the patient began to walk with a steppage gait.

Laboratory examination showed normal urine and spinal fluid; the Wassermann reaction of the blood was negative.

The temperature ranged from 97.6 to 101.4 for two days; then between 99.6 and normal for a few days; during the last two weeks of the stay in hospital it was normal. The pulse rate ranged from 64 to 100; the respiratory rate was normal.

Comment.—The case described combines the features of a central spinal neuritis and a peripheral neuritis, both due to an unknown toxic infectious cause entering by way of the respiratory system. The involvement of the cord produced a sensory level in the mid-dorsal region, a Babinski toe reflex (which came to light only as the function in the peripheral motor pathway was resumed) and retention of urine. The sensory losses in the glove and stocking areas were ascribable to involvement of the peripheral nerves. The motor phenomena may be laid at the door of involvement of either the central spinal or the peripheral nerves, except that the diminished right grip was, in all likelihood, of peripheral origin.

CASE 2.—*Secondary syphilitic infection of the lumbar cord with involvement of the anterior horn cells and pyramidal tracts, which was regarded as a septic, discrete myelitis for several months; in addition, an acute multiple arthritis, myositis and tenosynovitis. Improvement with the institution of antisyphilitic therapy.*

History.—J. B., married, aged 22, a housewife, entered Bellevue Hospital (neurologic service), Sept. 20, 1926. She was delivered of a child on July 21, 1926, following a dry labor. About ten days after delivery she began to have weakness and pains in both legs. On attempting to stand, she experienced cramps in the calves. Within a day, the pains spread upward to the thighs. Swelling of the feet and legs, which did not subside with rest, appeared. Five weeks later, pains in the arms, hands and elbow joints set in, being worse on the right than on the left. A week later, dysuria and frequency of urination developed. The pains increased and spread to the muscular tissues of the arms, forearms, wrists and small interphalangeal joints. In the lower extremities, the calves and thighs were tender and the ankles were swollen and painful.

Examination.—The patient was bedridden, pale and emaciated and looked ill and uncomfortable. The function of the cranial nerves was entirely normal. The upper

extremities were held flexed at the elbow; these joints, the wrists and the small joints of the hands and fingers were swollen and painful. In addition, the muscles and tendons of the left forearm were tender. Bitter complaint was made whenever any part of the upper extremities was moved about, so that tests of motor power could not be carried out satisfactorily. Focal atrophy or weakness was not found; the demonstrable atrophy was diffuse and could be interpreted as "reflex" to the arthritis, or due to disuse. The lower extremities showed marked foot-drop, with slight contractures at the ankle joints. Paralysis and atrophy were present in the anterior tibial and peroneal groups in both legs; the long flexors of the toes were greatly involved, the right more than the left, as were the intrinsic muscles of the feet. The calves possessed fairly good power. There was no weakness in the thighs. The abdominal musculature was normal.

The reflexes of the upper extremities were equal and active. The Hoffmann reflex was not elicited. The abdominal reflexes were lively and equal. In the lower extremities, the knee reflexes were active and equal; bilateral ankle clonus was present. The Babinski toe reflex could not be obtained because of paralysis in the long extensors of the big toes. However, positive Mendel-Bechterew and Rossolimo reflexes were elicited on the left.

The sensory status was normal, except for an inconstant hypalgesia of the distal parts of the extremities.

There were no signs of cerebellar disturbance.

At the laboratory examination, Sept. 26, 1926, the spinal fluid was clear and did not contain any cells; there was a normal amount of globulin, the colloidal gold curve was "flat," and the Wassermann reaction was negative. Two Wassermann tests of the blood were negative at this time. Urinalyses showed casts and a faint trace of albumin.

On Sept. 22, 1926, the white blood cells numbered 8,000, with 70 per cent polymorphonuclears; 18 per cent of lymphocytes, 7 per cent of large mononuclears and 5 per cent of transitional cells.

On Oct. 3, 1926, the red blood cells numbered 3,000,000, with 55 per cent of hemoglobin.

On Oct. 20, 1926, the red blood cells numbered 3,300,000, with 48 per cent of hemoglobin. Several blood cultures were negative. At this time, Wassermann tests of the blood were negative. From Sept. 26, 1926, to March, 1927, a "septic" fever persisted, the temperature ranging from 99 to 104. From March 15 to March 30, 1927, the temperature ranged from 99 to 100.

Course.—In an attempt to find a suspected focus of infection, the tonsils, teeth and sinuses and the genital, urinary and cardiorespiratory systems were thoroughly investigated. Some infected teeth were extracted; this was prompted by the development of an acute swelling of the lower part of the left side of the jaw, which subsided after the removal of the infected molars. Examination of the urinary system revealed a mild right pyelitis.

Salicylate and alkaline therapy were thoroughly exhibited without effect. In late November, 1926, a transfusion of blood was given.

The usual physiotherapeutic measures did not bring relief, and the patient remained ill and in constant distress. Help came from an unexpected source. Her infant became ill. The pediatrician suspected congenital syphilis; the Wassermann reaction of the child's blood was 4 plus; the husband's reacton was then found to be 4 plus, and on Jan. 5, 1927, the Wassermann reaction of the patient's blood, previously negative on several occasions, was 4 plus.

Antisyphilitic therapy was cautiously instituted, and gradual improvement set in. Eight intravenous injections of arsphenamine were given in twelve weeks;

for a time, iodide also was given. On March 30, the status was as follows: Innervation of the cranial nerves was normal. A mild bilateral temporomandibular arthritis was present, and a swollen lymph gland was felt in front of the left parotid gland. The upper extremities were weak and emaciated throughout. Fair power was retained in the deltoid muscles. All of the arthritic changes disappeared, except for mild arthritis in the small joints of the fingers and hands. There were flexor contractures at both elbows, more on the left than on the right. No trophic phenomena were found. The lower extremities showed marked wasting. The toes could be moved; the paralysis of the anterior tibial and peroneal groups was not so marked as previously, and the patient could produce dorsiflexion of both feet, more in the right than in the left foot. The strength in the calves was fair; it was greater in the left than in the right. The hamstrings showed fair power. The adductors and abductors of the thighs were fairly strong. There was bilateral weakness of the quadriceps extensors (knee) and of the flexors and extensors of the hips. There were contractures of both achilles tendons, more on the left than on the right, and of the hamstrings, more on the right than on the left. Trophic phenomena were not noted.

The ankle and knee reflexes were absent; the Babinski sign of the toe or confirmatory signs were not present. The left Mendel-Bechterew and Rossolimo signs previously noted had disappeared. The abdominal reflexes were active and equal. The biceps and triceps reflexes were active and equal. The Hoffmann sign was not present.

Sensory changes were absent.

The status at the later examination offered a marked contrast to that noted seven months before. The color was much improved; the appetite was good; the patient was comfortable and felt "convalescent." The syndrome then presented the features of a chronic lumbosacral anterior poliomyelitis.

Comment.—The case presented shows involvement of the anterior horn cells in the marked atrophy of the anterior tibial and peroneal muscle groups. The involvement of the pyramidal tract is shown by the bilateral ankle clonus and the left Mendel-Bechterew and Rossolimo signs. In the presence of such marked weakness of the lower motor neurons, the active knee reflexes also pointed to interruption of the pyramidal tract. In its neurologic aspect, we would call this a central spinal neuritis of syphilitic origin affecting the lumbosacral cord, terminating in a chronic anterior poliomyelitis. The only etiologic factor disclosed was syphilis. Another infection may have participated but, despite careful search, it was never brought to light.

In his monograph, Harris [11] mentions cases of this kind, saying that

"syphilitic infection occasionally produces generalized polyneuritis, though this is a rare complication. According to Pétren, true syphilitic polyneuritis occurs only within the first year after infection.

In some of the reported cases the neuritis appears to be associated with anterior horn cell damage as atrophy of certain groups of muscles such as the anterior tibial remained permanent.

11. Harris Wilfred: Neuritis and Neuralgia, New York, Oxford University Press, 1926.

In the foregoing case, disease of the anterior horn cells involved only the cell groups mentioned by Harris.

CASE 3.—*Ascending central spinal neuritis due to alcohol in a man with cerebrospinal syphilis. Recovery, with a final syndrome resembling cervical tabes dorsalis.*

History.—C. G., a Russian laborer, aged 40, was admitted to Bellevue Hospital (neurologic service), Nov. 11, 1926, complaining of paralysis of the lower extremities, weakness of the upper extremities, urinary incontinence and failing vision. The ascertainable family history, habits and past history were irrelevant except for chronic alcoholism. The present illness began suddenly on Sept. 24, 1926. He drank a great deal of vodka; the next day he felt ill and could not eat. On September 26, the lower extremities became so weak that he could not walk, and he could not urinate. Difficulty in swallowing liquid and solid food also appeared. On November 8, urinary incontinence was added to retention, and weakness in the hands developed. The patient made conflicting statements as to the date of onset of the visual difficulty.

Examination.—On Nov. 14, 1926, the man was acutely ill, emaciated, apathetic and bedridden.

The pupils were equal, irregular and fixed to light, but reacted slightly in accommodation. The disks were distinctly pale (primary optic atrophy). Bilateral ptosis was present. Sensation of the face and both corneas was normal. When the lower jaw was held open against resistance, it deviated to the right. The facial movements were normal. The left side of the tongue showed a patchy atrophy. The soft palate was weak on the left.

In the upper extremities, the grip of both hands was weak; the interossei were somewhat atrophied. No other weakness or atrophy was noted. The breathing was almost entirely abdominal. The lower extremities showed a complete flaccid paralysis with wasting of the musculature of the thigh, more on the right than on the left. Fibrillations were seen in the right calf.

The biceps reflexes were equal; the triceps reflexes were not elicited. The abdominal reflexes were absent, as were also the knee and ankle reflexes. The Babinski toe sign was not elicited. There was retention and incontinence of urine.

Sensory examination showed hyperalgesia over the lower extremities and tenderness of the calves on pressure. There was no definite disturbance in sensation to touch or joint position; vibratory sense was somewhat diminished in the lower extremities.

On November 17, the following additional symptoms were noted: In the forearms, the flexors were weaker than the extensors; weakness of the triceps was greater than that of the biceps; both deltoids were weak. The right latissimus dorsi was weaker than the left. The pharyngeal reflexes were absent; the palate moved poorly, the left side less than the right. Almost no movement was possible at the hips or knees, and slight dorsiflexion and extension at the ankles and toes. The sensory status was unchanged. The Wassermann reaction of the blood on November 10 was 3 plus. The cerebrospinal fluid showed 27 cells, an excess amount of globulin and a 4 plus Wassermann reaction. The urine was normal.

Course.—By December 12, vision was totally lost in the right eye, and only light and shadow were perceived on the left.

On Jan. 6, 1927, the man had become totally blind; the pupils were inactive, and bilateral ptosis was still present. There was a return of voluntary movement in the lower extremities and some improvement in the upper extremities. The

grip of the right hand was better than that of the left, and the small muscles of the hand were still atrophied; no fibrillations were present. The flaccid paraplegia had entirely disappeared, though the patient was still incontinent. The knee and ankle reflexes were normal. The biceps and triceps reflexes were absent. The Babinski sign of the toe and confirmatory signs were not present. The left abdominal reflexes were diminished and easily exhausted; the right were absent. The calves and achilles tendons were exquisitely tender to pressure. There were no other objective sensory symptoms. This residual status resembled cervical tabes dorsalis.

Comment.—The flaccid paraplegia with loss of reflexes, the disturbance of the bladder, the rapid ascent under observation, with subsequent involvement of the upper extremities, and weakness of the intercostal muscles, tongue and palate bespeak an acute ascending spinal neuritis reminiscent of Landry's paralysis. It is interesting to note that as early as 1896 Diller and Meyer [12] stressed the implication of both peripheral and central nervous systems in a review of the reported cases of Landry's paralysis. This diagnosis was made by a member of the staff before an adequate history was obtained. The fixed pupils and the primary atrophy of the optic nerve suggest cerebrospinal syphilis, which was confirmed by the positive serologic symptoms. It must be admitted that wood alcohol may have accounted for the optic atrophy, but the pupils were fixed to light while vision was still fair, a fact pointing to the syphilitic nature of the pupillary paralysis. Perhaps the hyperalgesia in the lower extremities and the tenderness in the calves were due to peripheral neuritis.

CASE 4.—*Toxic alcoholic lumbosacral radiculitis with evidence of slight involvement of the spinal cord.*

History.—T. C., an Irish laborer, aged 39, entered Bellevue Hospital (neurologic service), July 28, 1926, complaining of a "dead feeling" in both feet. The past history was unimportant, except for marked alcoholism of three or more years' duration. He had a congenital right internal strabismus and had been deaf in the left ear for ten years. He stated that he had lost 40 or 50 pounds (18.1 or 22.7 Kg.) within the year preceding admission to the hospital. The present illness began in the latter part of 1925, with a "dead feeling" in both feet, because of which he entered the medical service of Bellevue Hospital in June, 1926. He was discharged after eight days, with a diagnosis of alcoholic gastritis.

In June, 1926, he complained of burning in the feet with weakness which caused difficulty in walking. He noted that he was unable to feel touch, heat and cold on the skin of both feet and lower legs, and that the strength of the hands and arms was less than formerly. At about this time he suffered from headache, anorexia, malaise, chills and "fever" for a few days. Pains and sweating in the feet and legs developed. The sensory symptoms spread up the lower extremities.

12. Diller, T., and Meyer, A.: A Case of Landry's Paralysis, with Autopsy, Am. J. M. Sc. **111**:404, 1896.

Neurologic Examination.—On Aug. 1, 1926, function of the cranial nerves was normal, except for a right internal strabismus and conduction deafness in the left ear. Both conditions were of many years' standing and antedated the present illness. Both the upper extremities were weak throughout. The gait showed a slightly spastic quality. Fibrillary twitchings were present in both legs, more noticeable on the right. There was atrophy of the quadriceps extensors, more marked on the right, and of the right hamstrings, with flattening of the right gluteal fold. The measurements were: left middle calf, 39 cm.; right middle calf, 38 cm.; left thigh, 49 cm.; right thigh, 46 cm. Bladder or rectal disturbances were not present.

The reflexes in the upper extremities were active and equal. The knee reflexes were active and equal; the right ankle reflex was absent, the left diminished; no Babinski toe reflex was elicited. The abdominal reflexes were absent, except for an exhaustible left upper reflex; the cremasteric reflexes were not elicited. A strong costal margin reflex was obtained on the left and a weaker one on the right.

Signs of cerebellar disturbance were not present.

Sensory examination revealed a level at about the eighth cervical dermatome, below which pain and touch were diminished. Another observer found this level at the third dorsal dermatome. A third examiner found a definite sensory defect from the first lumbar to the first sacral dermatomes. Within this area were diminution or loss of pain, diminution of touch, marked decrease of appreciation of temperature, and diminution or loss of vibratory sense. A less marked involvement of the remaining sacral dermatomes was noted. Sensation to joint position and deep pain were retained. Marked sweating of the feet was present.

Manometric examination of the spinal fluid showed an initial normal level at 80 mm., with a barely perceptible rise on touch compression of the jugular veins. On deep compression, the fluid rose to 170 mm. on one occasion, and to 200 mm. on another; it was more than thirty seconds before it returned to normal. Straining caused a prompt rise to 240, with a fairly prompt fall. After removal of 7.5 cc. of spinal fluid, the pressure fell from 80 to 30 mm., and on deep compression rose from 30 to 140 mm. The result of the manometric examination was indefinite and not conclusive. The spinal fluid was normal.

Comment.—This case combines a lumbosacral radiculitis with involvement of the cord. The sensory changes in typical dermatome areas point to disease of the roots. We ascribe the loss of the abdominal reflexes, the unilateral increase of the left periosteal costal margin reflex, the fibrillations and the sensory levels on the trunk to the involvement of the cord. These were definite enough to make two observers suspect tumor of the spinal cord. We would call such a syndrome a lumbosacral radiculitis with central spinal neuritis.

CASE 5.—*An obscure infection of the peripheral nerves, roots, meninges and cord. Course punctuated by exacerbations; a slow stormy convalescence ending in complete recovery.*

History.—L. E., a German governess, single, aged 27, entered Bellevue Hospital (neurologic service), March 23, 1926, complaining of a "hot" tingling, alternating with coldness and numbness in the forearms, legs and feet, of three weeks' duration. The past history, family history and habits were irrelevant.

On Jan. 6, 1926, she fell into an excavation and got wet from the hips down. She then walked in the cold night air for two hours. There were no immediate consequences. The present illness began suddenly on March 2, 1926, three weeks before admission to the hospital.

The first thing she noticed was that on arising from a sitting posture she felt a sensation of numbness and "deadness" in the feet and ankles, to which was added a feeling of heat, tingling and swelling ("as if one were thawing out one's hands and feet"). This paresthesia spread upward to the waist-line and appeared in the upper extremities. She also complained of intense generalized weariness and a deep aching in the lower extremities, backache and pronounced frontal headache. Clumsiness in the hands interfered with writing and other fine movements. There had been a recent anorexia and loss of weight. Blurring of vision had been present for an indefinite period.

Neurologic Examination.—On March 25, 1926, the function of the cranial nerves was normal, except for blurring of the nasal margins of both disks and congestion of the retinal veins. The upper extremities showed normal power. The lower extremities were weak, the right more so than the left; there was dragging of the right lower extremity in walking.

The biceps, radial and triceps reflexes were active and equal. The abdominal reflexes were active, but diminished on the left. Bilateral patellar clonus, hyperactive achilles reflexes and bilateral Babinski toe sign, the last more marked on the right, were elicited.

Signs of cerebellar disturbance were not present.

Hyperesthesias and paresthesias of glove distribution in the upper extremities up to the elbows were present. On the trunk a poorly defined sensory level was demonstrated at about the sixth dorsal dermatome, below which all forms of sensation were diminished; this was less evident over the low sacral areas. A girdle sensation was complained of in the area between the sixth and eighth dorsal dermatomes. The mid-dorsal spine (from the fifth to the eighth) was tender. Disturbances of the sphincters were not present.

On March 26, examination of the spinal fluid disclosed: a colorless, slightly cloudy fluid; increased globulin (2 plus); 500 cells (85 per cent lymphocytes); colloidal gold curve, 5554321100. The systolic blood pressure was 95; diastolic, 70. Roentgenograms of the vertebral column were normal. The temperature was normal.

Reexamination, April 8, showed the following changes: The patient walked insecurely, due to weakness, with a poor associated swing of both upper extremities. At the third dorsal level, there was a band of increased sensitivity to touch, pain and temperature. The scalp (second and third cervical levels) seemed hypesthetic. Below the fifth dorsal level the diminution to all forms of sensation persisted, except joint position and vibratory sense, which were normal. Both hands and foreams showed a glove area of hypesthesia and hypalgesia. This defect shaded gradually into normal sensibility proximally.

Course.—Convalescence was gradual, but often interrupted. Dizziness, restlessness, headache, nausea and vomiting and pains in the right lower extremity, legs and back would occur in attacks of several days' duration, during which the temperature ranged from 99 to 101 F. In these episodes, there would be various combinations of these symptoms.

On April 23, 1926, reexamination of the cerebrospinal fluid showed: normal pressure; clear color; 20 cells; globulin, 2 plus; a negative Wassermann reaction; no growth on culture, and a colloidal gold curve of 3332211000. Typhoid vaccine

was given intravenously, beginning April 30. It was continued, though the patient complained of pain in the back and legs and stiffness in the upper and lower extremities during the period of its administration.

On June 1, 1926, the patient was reported as doing well; she would sit in the sun parlor for a few hours each day without undue fatigue, and was apparently gaining strength. The day before this, however, she felt fatigued and slept poorly during the night because of a severe, dull, heavy pressure headache. The fatigue increased, and "general body heaviness" became intolerable. Numbness, tingling and mild cramps developed in the hands and forearms, with a tendency to flexor contractures. Except for the cramps, similar sensations appeared in the trunk and lower extremities; a painful tingling ran up and down the spinal column. There was no difficulty in control of the bladder.

Examination disclosed normal function of the cranial nerves, except for a subjective numbness on the entire left side of the face and neck, and "heaviness" on the left side of the tongue. The defect in sensation extended to a line running down on the left of the midline. No objective signs were found. The corneal and palatal reflexes were present.

There was general weakness in the extremities, more marked in the legs than in the arms.

The deep reflexes were everywhere active and equal. The abdominal and epigastric reflexes were absent. The plantar response showed normal flexion. Sensory examination disclosed the same hypesthesia of the scalp as revealed at the previous examination and a level at the eighth dorsal segment, below which sensation was diminished; appreciation of heat and cold gradually decreased and was lost below the knees. Joint position sense was not affected, while vibratory sense was slightly dulled, more on the left side. The ulnar areas of both hands and forearms felt numb. Areas of distribution of the peripheral nerves were said to be hypesthetic or hyperesthetic in the lower extremities. No astereognosis could be demonstrated. The patient complained bitterly of a girdle sensation. Pressure of the calves, thighs and arms was painful, more especially on the right. Ataxia was not present.

At the time of this exacerbation, the temperature ranged from 97 to 101; the pulse rate from 70 to 120 and the respiratory rate from 18 to 24. The blood showed 10,000 white cells, with 78 per cent polymorphonuclears; 20 per cent lymphocytes and 2 per cent transitional cells. The urine showed many squamous epithelial cells, pus cells, red cells and mucus.

The patient finally made a complete recovery after several months.

Comment.—The acute infectious process involved the peripheral nerves, as evidenced by typical glove areas of sensory disturbance in the upper extremities, peripheral fields in the lower extremities and tender nerve trunks. The girdle sensation and the various pains and paresthesias may be ascribed to involvement of the roots, the peripheral nerves or to both. The areas of abnormal sensation on the trunk, the double patellar clonus, hyperactive achilles reflexes and double Babinski signs in the toes bespeak involvement of the spinal cord. The tender, painful spine and the manifestations in the spinal fluid, more especially the high cell count, point to meningeal involvement, which could also explain the headache and the mild papilledema. The gradual but stormy convalescence is of especial interest. No clue as to the nature of the causative agent was found.

SUMMARY

1. A series of five nonfatal cases is presented, illustrating toxic or infective processes involving the peripheral nerves and the central neuraxis. This motley group, with a varied etiology, shows a mixed symptomatology referable to the peripheral and central nervous system.

2. A simple nomenclature is suggested in which the term neuritis is retained—peripheral neuritis for processes limited to the distal parts of the nerve trunks; radiculitis, for involvement of the roots, and central neuritis (spinal, medullary, pontile, etc.) for involvement of the neuraxis. When more than one part of the nervous system is implicated, these terms may easily be compounded.

News and Comment

THE BUDAPEST CONGRESS

The Fifth International Medical Congress for Industrial Accidents and Occupational Diseases is to be held in Budapest during September, 1928.

A National Committee for the United States has been created with Dr. Emery R. Hayhurst, Hamilton Hall, Ohio State University, Columbus, Ohio, Chairman. Addresses are already scheduled by prominent Europeans. Addresses and lectures are wanted from American physicians, who are requested to get in touch with the chairman.

Abstracts from Current Literature

CEREBRAL AND CEREBELLAR NEUROGLIA IN GENERAL PROGRESSIVE PARALYSIS, WITH SOME TECHNICAL INDICATIONS ON SILVER IMPREGNATION OF PATHOLOGIC NERVE TISSUE. S. R. CAJAL, Trav. du lab. de recherches biol. de l'univ. de Madrid **23**:157 (Nov.) 1925.

The material for this study was taken from advanced forms of progressive paralysis in adults and old patients. The cortical areas studied in detail were: the ascending central convolution (number 4 of Brodmann); the fissure of Sylvius; the ascending parietal convolution (regio-postcentralis of Brodmann); the calcarine fissure; the occipital convolutions in the vicinity of the last, and the cornu ammonis. The cerebellum also was studied.

In addition to the classic methods of Nissl, Weigert, Alzheimer, Da Fano, Mallory, Held, Spielmeyer (for myelin), Achucarro, Del Rio Hortega, et al., the following procedures, modifications of methods already published, were used:

I. For Staining the Macroglia

1. Immerse frozen sections (from 25 to 35 microns) of material preserved in formaldehyde-ammonium bromide (from two to twenty-five days or even for a month) for four hours at room temperature or, still better, in the thermostat, in the following "reenforcing" solution:

Ammonium bromide	3 Gm.
Neutral formaldehyde, 40 per cent	30 cc.
Water	70 cc.

2. Quickly wash sections (a few seconds) in three changes of water, then transfer to:

Normal solution of silver-ammonia (Bielschowsky)	10 cc.
Pyridine	7 to 10 drops
Water	10 to 12 cc.

After from five to ten minutes at room temperature, place a Stender dish with the sections over a flame until they acquire a brownish color.

3. Wash quickly in two changes of distilled water and reduce in 5 per cent neutral formaldehyde (40 per cent).

4. Tone with gold chloride, fix in sodium thiosulphate, wash, dehydrate and follow by oil of clove, xylene and dammar.

II. For Staining the Microglia

Cajal used the method described, with the following modifications:

1. Immerse sections in the silver solution until they show a light straw color (from five to ten minutes at room temperature).

2. Warm gently over a lamp until the sections acquire a yellow and slightly brownish tone. If this color has already appeared in the cold solution, heating can be omitted.

3. Wash quickly (from three to five seconds) in two changes of water.

4. Tone with gold chloride (from five to ten minutes at room temperature), and fix in sodium thiosulphate. If the reaction was successful, sections appeared light purple.

III. For Nerve Fibers

1. Wash frozen sections (from 30 to 40 microns) of material preserved in 14 per cent liquor formaldehydi in two changes of water.

2. Transfer (five or more minutes at room temperature) to:

2 per cent aqueous solution of silver nitrate	10 cc.
Pyridine	8 drops

3. Warm gently until sections appear light brown.

4. Before sections are cool, transfer to 96 per cent alcohol (1 to 3 seconds).

5. Without previous washing reduce in:

Hydrochinone	0.20 to 0.30	Gm.
Distilled water	70	cc.
Neutral formaldehyde, 40 per cent	30	cc.

The reduction is complete in about one minute.

6. Toning with gold chloride is not necessary, but if desired, immerse sections, after washing, in 1:300 solution of yellow gold chloride. Fix with sodium thiosulphate, wash again. Dehydrate, etc.

The author agrees with most investigators who have studied the problem that the chief characteristics of the disease in the cerebrum are a considerable increase and hypertrophy of the macroglia and the transformation of the protoplasmic glia of the gray matter into fibrous glia. These processes coexist with destruction of the glia in certain places, accompanied by proliferation of the cells in the vicinity. Formative centers consisting of several young glia cells are not uncommon, and such masses undoubtedly correspond to the Rasen of Nissl. Eventually, there occur sclerotic foci formed by large masses of the fibers of Weigert, and areas of softening in which the implantation feet of the glia cells adopt a converging orientation around totally or partially destroyed vessels. On the other hand, stout bundles forming anastomoses between the vessels exist; these structures resemble the cords of Robertson, but are composed of fibers of Weigert, as shown by their staining reactions. The formation of a stout subpial plexus of fibers of Weigert is also an almost constant feature in the cerebrum. It is not unusual to find in the lower part of the molecular layer giant macroglia cells forming wreaths of thick, descending fibers. Perivascular infiltrations were abundant in the slides studied.

As shown by Nissl and his students, the rod-shaped microglia (Stäbchenzellen) appear enlarged and much thickened. The stellate microglia are also considerably increased in volume and exhibit a smoother and simpler appearance than in sections from normal brains. Some of the stellate microglia cells acquire an enormous size and appear chiefly localized in the vicinity of the vessels, where they adopt extremely unusual shapes. Microglia cells were never found inside the vessels or in the infiltrations and mesodermic nodules. This leads to the belief that the rod and stellate cells represent, for the most part, preexisting elements endowed with migratory capacity which move toward the neurons in search of products of degeneration. The opinions of Nissl, Alzheimer, Achucarro and Gayarre, Spielmeyer, Del Rio Hortega and other investigators are thus confirmed. The rod shape so common in the microglia in general progressive paralysis is explained as the result of a double adaptation of the cells to the migratory function, and to the spaces between the nervous structures, a view already expressed by Achucarro. The adaptation just mentioned is regarded as a phenomenon of stereotropism similar to that occurring in the growing embryonic fibers and in the stumps of regenerating

nerves. Finally, medullated nerve fibers are relatively little affected in general paralysis in the cerebrum, and amedullated fibers are still less affected. There occur, however, zones in which fibers are scarce; fragmented axons and round masses are produced through this fragmentation. Such lesions are particularly common in the white matter. Lesions of the neurons were also found and are briefly described, since they have already been reported with detail by many authors.

The characteristic lesion of the cerebellum is gliosis, accompanied by degeneration and partial destruction of the cells of Purkinje and a decrease in the number of granules and baskets. Gliosis is most pronounced in the molecular layer, in which a plexus of the fibers of Weigert occurs. The components of the plexus have at least two origins: (*a*) they arise through fibrous transformation of the prolongations of Bergmann and of their cells, and (*b*) through penetration of fibers arising from the glia of the granular layer and the white matter into the molecular layer. The opinion of several authors on the existence of proliferation of the epithelial cells is confirmed. The proliferation is related to the degree of destruction of the cells of Purkinje, as affirmed by some investigators.

The microglia of the molecular layer of the cerebellum are abundant and show marked hypertrophy; many of the spines and secondary processes of the cells disappear. The origin of these cells in the pia or in the vessels is doubtful. Giant macroglia cells with several nuclei and abundant processes occur in degenerated areas of the white matter. The glia cells of the granular layer often adopt fibrous types, but they do not increase much in diameter.

In certain cerebellar regions both climbing and parallel fibers are absent, and transverse conductors are scarce. Medullated fibers are not much affected by the morbid process. The most characteristic lesion in the granular layer is the presence of mossy fibers with collateral fenestrated branches and giant endings, more conspicuous in areas in which the morbid changes are just beginning.

The much discussed problem of the precedence of vascular disorders or nervous disorders in general paralysis could not be solved since the material used was from adult or old subjects with advanced lesions. Cajal agrees with Spielmeyer and his school that all that can be said in this connection is that in both the cerebrum and the cerebellum areas exist in which vascular disorders are slight and nervous and glial disorders are pronounced, and vice versa.

NONIDEZ, New York.

FRACTURES OF THE SKULL. B. M. VANCE, Arch. Surg. **14**:1023 (May) 1927.

Vance has carefully analyzed 512 cases of fracture of the skull verified at autopsy. He classifies the fractures into those of the vault and base and those of the vault alone. It is striking to realize that in 471 cases both vault and base were involved and that only in thirty-six the vault alone was involved, showing definitely the tendency of the fracture line to travel downward. A brief discussion of the mechanism of fractures is given, and the various types are well illustrated by diagrams. As was to be expected, automobile accidents predominated as the causative factor in producing the injury.

Concussion is recognized as a clinical entity and given as the cause of death in 139 cases. Almost all of these patients succumbed in the first ten hours after injury. It should be noted, however, that accompanying bruises or severe lacerations of the brain were entirely absent in only sixteen instances. The casualties that produced the concussion and subsequent rapid death were in

general of the more severe type. In this group, fractures of the posterior part of the skull were relatively numerous. Interestingly enough, alcohol was found by chemical analysis in the brain and liver of forty of these 139 cases, a ratio of 28.6 per cent. Alcohol not only increases the chance of injury by dulling the faculties and thus rendering the individual more prone to become involved in an accident, but furthermore, by its depressing effect on vital nerve centers, it makes diagnosis difficult. By lowering the patient's resistance, alcohol makes a fatal result more likely to follow a moderate degree of violence. Besides concussion, the more frequent causes of death in this series were cerebral compression, lacerations of the brain, and subdural hemorrhage (156 cases), and cerebral compression following extradural hemorrhage (61 cases). Forty-eight patients died from meningitis and other septic conditions.

The majority of subdural hemorrhages were the result of contre-coup or direct lacerations of the cerebral cortex, especially over the frontal lobes following a laceration of the brain at the tips of these lobes. A few were produced by the tearing of tributary veins from the brain to the superior longitudinal sinus, and one was caused by the tearing of an old adhesion between the brain and the dura. In one case, laceration of the dura and a branch of the middle meningeal artery caused the artery to bleed profusely into the right subdural space.

The fatal lacerations of the brain were either surface lesions which covered a wide area of the cerebral cortex or big cavities in the brain substance which contained a large quantity of blood. Some of these caused extensive hemorrhages in the ventricles of the brain. None of the lesions, however, were accompanied by a subdural hemorrhage of any size. There was reason to believe that many of the cases in this group showed a moderate grade of edema of the brain.

Sixty-one patients died of cerebral compression caused by an epidural hemorrhage. Most of the hemorrhages were the result of a laceration of the middle meningeal artery by the fractured bone. The posterior branches of the middle meningeal artery were involved much more frequently than the anterior branches. A few hemorrhages were caused by lacerations of the lateral sinus. One hemorrhage arose from a laceration of one of the anterior meningeal arteries.

The interval between the casualty and the death varied considerably in the cases of cerebral compression. Most persons succumbed during the first twenty-four hours, but others lingered for from eight to fourteen days.

In connection with contre-coup injuries, it was found definitely that these only occurred with the head in active motion as when a patient was thrown and struck the head against some solid surface. Vance adheres to the theory that it is the sudden arrest of the brain within its bony box, so that it is thrown violently against the opposite side of the vault from the point of impact, that causes this type of injury. It was never seen when the skull was subjected to trauma of the slow crushing type. Furthermore, these contre-coup injuries are most commonly encountered on the under and outer surfaces of the frontal and temporosphenoidal lobes in which the brain rests most closely on the dura and in which the impact from the aftercoming bone could be most easily and directly transmitted to it.

The second part of the paper takes up the clinical aspect of complications produced by fractures of the skull. The causes are divided into: (1) fatal cases in which craniotomy was not performed; (2) operative cases, fatal and non-fatal; (3) fractures of the skull from which the patients recovered under expectant treatment.

In deciding on the advisability of operation, the clinician must answer two questions: 1. Do the clinical observations indicate a fracture of the skull? 2. Are the signs definite enough to identify an intracranial condition in which benefit will be derived from craniotomy? Sixty-one cases are considered in detail. Thirty-four patients were operated on; eleven recovered. The indications for operation and the results were as follows: (*a*) compound or depressed fractures — fatal cases, three; recovery, seven; total, ten; (*b*) increased intracranial pressure — fatal cases, twenty; recovery, three; total, twenty-three; (*c*) local paralyses — fatal cases 0; recovery, one; total, one.

The fact on which the diagnosis of fracture and indications for operation were based are considered at length: a history of trauma or evidence of external injury; the general condition of the patient; pulse, respiration and blood pressure readings; lumbar puncture pressure; ophthalmologic studies (which curiously enough Vance found of value), and the neurologic symptoms. The reader is referred to the original paper for these details.

In commenting on the series, Vance says: "The sixty-one cases considered showed a preponderance of fractures in the lateral part of the skull, with relatively few located anteriorly and posteriorly. They were found as follows: lateral fractures, forty-eight; anterior fractures, seven; posterior fractures, six. The reason for this disproportion was probably that the lateral variety was more easily discovered by the roentgen ray than the other types; and also that this variety was accompanied by such characteristic external lesions as large hemorrhages of the scalp or bleeding from the canal of the external ear. In addition, lateral fractures usually involved the motor cortex in various ways and thus produced striking neurologic signs, while anterior and posterior fractures, even when severe, tended to involve the "silent" areas of the brain and only occasionally elicited a definite neurologic phenomenon.

"In general, with the exception of such outspoken indications as compound or depressed fractures, patients with the anterior and posterior group of lesions did not require operation in the normal course of events. These seemed either to cause complications which were almost inevitably fatal or else to be accompanied by such slight intracranial lesions that the patient recovered spontaneously.

"Lateral fractures displayed these same characteristics to a certain extent, but at the same time produced a large number of intracranial injuries which were amenable to a craniotomy, but which invariably proved fatal if left alone. Accordingly, lateral fractures formed the vast majority of those in patients subjected to craniotomy."

It is interesting to note that in a summary of his follow-up records, Vance states definitely that the eleven patients recovering after operation showed a much greater variety of unfortunate sequelae and after-effects than did the twenty-seven patients who recovered under expectant treatment. It is possible, of course, that the former group on the whole had a more severe degree of injury than the latter, but, nevertheless, such a statement from a careful observer should carry weight. This is a valuable piece of work and merits careful study.

GRANT, Philadelphia.

CAUSAL AND COMPREHENSIBLE ASSOCIATIONS IN EPILEPSY. HANS BINDER, Schweiz. Arch. f. Neurol. u. Psychiat. **19**:242, 1926.

Hans Binder reports a case of epilepsy which he studied with great care. The history is essentially as follows: A sister of the patient had had typical and frequent attacks of epilepsy while she was in the twenties, which disappeared after several years. The father had had a severe form of epilepsy. In his

fortieth year, he had developed recurrent attacks of irritability which lasted several days. These had become progressively worse, and during these times he had often beaten his wife and children on the slightest provocation, which he had never done at other times. About three years later, he had disappeared for periods of from two to four days. After his return from these flights, he had been unable to say where he had been. He also had had attacks of confusion, in which he would run about the house, speak in a confused manner, believe that he was being pursued, nail his clothing to the wall, thinking that he was papering the wall, and throw food into the stove for fuel. Six years after the onset, the first epileptiform convulsions had appeared. He had threatened members of the family with an ax, following which he had been committed to an institution. Twelve years after the onset he had become demented, and sixteen years after had died in a status epilepticus.

The patient himself had been greatly attached to his mother; he had been faithful and industrious, and had done well in school as a child. He had been exceedingly afraid of the dark. During the war he fell from a horse, following which he was unconscious for a day. Later he suffered from an attack of otitis media. At the age of 21, he returned to the service and was gassed, following which he was unconscious for eight days. He was admitted to the Heidelberg clinic at the age of 31. At 22 years of age, one year after he was gassed, he developed dream states and periods of marked irritability. These occurred every month and lasted from two to five days. Without any known cause he would suddenly become fearful and irritable, losing his temper when he saw a fly on the wall and picking a quarrel with every one. He complained of headache, dizziness, scotoma and nausea and usually remained in bed.

The "Dämmerzustände" were somewhat as follows: He would suddenly feel a marked pressure which would urge him to flee. For the first few hours he could recognize the streets through which he passed, but later could recall nothing. On recovering from one attack, he remembered that he was feeling about and discovered that he was sitting on a bench. During this time, he could see only a small circular field. This enlarged, and he was again able to see and think. He inquired the name of the city he was in and proceeded home, begging on the way. He also suffered from sudden attacks of intense fear and felt as though a policeman were near him. The policeman resembled his father. There were also attacks of petit mal lasting ten seconds, during which the head would suddenly droop, saliva run out of the mouth, and the pupils enlarge and become fixed. He also had attacks of grand mal. Since the onset of the illness, he became drunk on a small amount of wine. He became very religious and emphasized his sympathetic nature. One day he suddenly told his wife about a position he had in America. He refused to go to work, did not eat, struck his wife, attacked neighbors with a knife and announced that he was going to another city to get money for the journey. Of all of this he could remember nothing. A number of details could be cleared up in the hypnotic state. He hated and feared his father. When he was 6 years old, his father had given him a cruel beating which his mother terminated. At 10 years of age, he had suddenly developed a fear that his father had died.

The resemblance of the patient's illness to that of the father is close. Binder thinks that the son inherited a disposition for the development of a particular type of epilepsy; this explains factors in the patient's illness that cannot be explained by his personal experiences. The father's attacks of irritation and fugues marked the initial stage of an organic brain process, which progressed to dementia and death. The son probably inherited the anlage for the development of a similar condition. Close investigation of the patient seemed to reveal

many psychopathic episodes in his early life. Periodic episodes did not begin until the age of 22. There is no psychologic reason why the patient should suddenly develop such disturbances at this age, and one is forced to assume that an organic process is responsible. Following its onset, the condition became progressively worse. It may be that the injury sustained in the war precipitated the appearance of these attacks. If it is assumed that these attacks are reactive, one must grant that an environmental occurrence took place every few months, which is unlikely. The attacks, furthermore, were almost similar. The sudden cessation of the attacks also argues for an organic basis. It was impossible to produce any of these attacks by hypnotic suggestion. The content of the fugues could not be cleared up entirely by hypnosis.

The father was an obstacle in the love for the mother and for a school girl friend. At the present time, however, his wife has taken over the rôle of the father, so that the homicidal fantasies have been transferred to her. This is clearly a case of regression. The homicidal tendencies, on the other hand, called forth still stronger inhibitory reactions; these led to the fugues. Without closer analysis one might have assumed that a psychogenic mechanism was responsible for the various symptoms. This explanation is not altogether satisfactory.

In the older literature the opinion was held that fugues are practically always an expression of epilepsy. Heilbronner, for the first time, emphasized that this was by no means always the case, and Stier, in 1913, came to the conclusion that only a small percentage of these cases are epileptic and that most of them are hysterical, psychopathic or schizophrenic. The diagnosis of epilepsy in this patient cannot be questioned. Schultz has emphasized that major epileptic attacks are, as a rule, infrequent in patients who exhibit fugues. Examination of the fugue itself cannot determine its nature; inspection of other factors usually does enable one to determine this. Organized movements and retained memories in themselves are not incompatible with the diagnosis of an epileptic fugue. It is generally conceded that all experiences preserved in the psyche are active in disturbances of consciousness even when these are due to organic lesions.

Kraepelin and Bleuler compare epileptic "Dämmerzustände" to dreams. Mäder, for the first time, was able to uncover the content of a prolonged "Dämmerzustand" through hypnosis in a patient who had alcoholic epilepsy. The emotional disorder occurring in epileptic patients often causes them to undertake fugues which they would not carry out otherwise. Plans made during normal periods may pursue their course during a "Dämmerzustand." It is then that they become compulsions. Mäder compares the content of epileptic "Dämmerzustände" with those of hysteria and explains them as a wish fulfilment, principally of a sexual type. Schilder believes that they are often dominated by an idea of death and rebirth. Stekel does not grant that any somatic cause enters into the etiology of genuine epilepsy, but regards these attacks as regressions into an embryonic existence. Epileptic "Dämmerzustände" generally exhibit similar psychic manifestations, whereas those of hysteria present varying pictures. The epileptic character, which overemphasizes kindness, evenness of temperment, tenderness, religious sentimentality and sweet moralizations, can be explained as an "Abwehrreaktion" from primitive hatreds.

WOLTMAN, Rochester, Minn.

TWO CASES OF HYPOPLASIA PONTONEOCEREBELLARIS. S. KOSTER, Acta psychiat. et neurol. **1**:47, 1926.

The author presents a pathologic study of two cases of hypoplasia of the pons and neocerebellum. His first case was that of a girl, who died from

diphtheria at the age of 1 year. Further clinical data were not available. The posterior fossa and its contents were very small. The cerebellum and the brain stem together weighed only 15 Gm. The cerebellar hemispheres appeared as two flattened disks, and this poor development left the anterior part of the vermis uncovered. Only a few primary convolutions were present, except in the lateral and ventral parts, where there were a few secondary lobuli, and dorsally and caudally, where a few secondary and tertiary lobuli could be distinguished. The parts that showed a greater degree of development corresponded to the lobus cuneiformis. The vermis, flocculi and tonsils, though small, were normal. Only a small portion of the pons was present. The pyramidal and olivary protuberances were of normal size.

Histologically, the satification of the cerebellar cortex was normal. The molecular and granular zones of the hemispheres were thin, and in the areas where this thinning occurred the number of Purkinje cells was less than normal. In the lateral part of the lobus anterior and lobus simplex the Purkinje cells were small and slender. The central white matter was so poorly developed that the nucleus dentatus was almost in touch with the gray matter of the cortex. The radiation of the middle cerebellar peduncles could scarcely be distinguished. The white matter of the vermis was normal. The convoluted form of the dentate nucleus had disappeared and the nucleus consisted of only small nests of cells surrounded by myelinized fibers. These cells were present only in the dorsomedial (paleocerebellar) part. The nucleus emboliformis and nucleus tecti were more nearly normal than was the dentate nucleus, but they were small. The nucleus globosus was normal. The superior and inferior cerebellar peduncles were diminished greatly in size, while the middle consisted only of a few fiber bundles.

The nuclei arcuati were small, and the cells were of embryologic type. Only a few cells of the corpus pontobulbare could be distinguished. With the exception of the pyramidal bundles, the basis pontis was nearly absent. The frontopontile fibers were normal as far as the region of the root fibers of the facial nerve, caudad to which they could not be found. The temporopontile tract was normal as far as the root fibers of the trigeminal nerve. A few fibers in the basis pontis represented the strata superficiale, profundum and complexum. Most of the cells of the pontile nuclei were absent, more so in the caudal than in the cephalad parts. The inferior olivary nuclei were small. The lateral portions of the olivary convolutions showed the most abnormalities, and, except at the caudal pole, the ventral portions were developed best. In the portions of the nucleus in which no cells were found, the tissue appeared undifferentiated. Only a thin olivary capsule was present. The caudal part of the red nucleus was small, and only large cells were found. The capsule was absent. The caudate nucleus was very large.

The second case was that of a child, aged 21 months, whose clinical history was unknown. A condition almost identical with that in the first case was found on pathologic examination, except that there was a heterotopia of Purkinje cells in one cerebellar tonsil, and the inferior olivary nuclei were better developed.

In discussing these cases the author points out that the pons, nuclei arcuati and inferior olivary nuclei develop from cells situated in the medulla, close to the lateral border of the fourth ventricle. From this area, the cells migrate in two directions, presumably under neurobiotactic influences. One group passes between the fibers of the seventh and eighth cranial nerves, lateral to the restiform body, in the direction of the frontoparietotemporopontile tracts, to form the cells of the pons. The other group migrates in a ventrocaudal direction to form the inferior olivary nucleus and the arcuate nuclei. The cerebellum

develops from a germinal center in the roof of the fourth ventricle contiguous to the area just described. Both Winkler and Brouwer consider that if a pathologic factor operates on these adjacent areas before the migration of the cells has begun the result will be a hypoplasia of the basis pontis, cerebellum and inferior olivary nuclei. The migration takes place earliest to the last mentioned nuclei, that to the pons occurring latest. In the author's cases the etiologic process must have taken effect after the migration to the olives was nearly complete but before that to the basis pontis, cerebellum and arcuate nuclei had commenced. This would be about the third fetal month.

Besides the injury to the germinal centers, he considers that that part of the hypoplasia, particularly that affecting the inferior olives, is the result of what he calls the correlative factor, i. e., an injury to one part of the developing nervous system which produced maldevelopment of those parts connected with it. In the instance of the hypoplasia of the basis pontis, Koster thinks the influence of the correlative factor has a bearing also, for the consensus of opinion is that the pontile nuclei send their axons mostly to the contralateral cerebellar hemisphere, although a few may go to the ipsilateral one, but none go to other parts of the brain.

The location of the hypoplasia follows the law that developmental defects implicate the newer structures most. From numerous phylogenetic and ontogenetic studies, the cerebellum has been divided into two portions, the paleocerebellum — the vermis, flocculus and paraflocculus — and the neocerebellum — the cerebellar hemispheres. Van Valkenburg considered that the lateral parts of the lobus anterior and a small part of the lobus simplex formed a transition zone between the new and the old structures. In Koster's cases the neocerebellum was hypoplastic, and the defects included these areas. It seems reasonable to conclude that they are really parts of the neocerebellum. In regard to the cerebellar nuclei, the ventrolateral-caudal portion of the dentate nucleus is a new structure; the remainder of this nucleus and the other nuclei are old. The former part shows marked hypoplasia. Also the hypoplasia of the inferior olivary nuclei follows the phylogenetic and ontogenetic development of these structures. The best preserved parts are the oldest, i. e., the medioventral and dorsal accessory olives. Brouwer is of the opinion that the oral part of the main nucleus was old also, but in Koster's cases this was hypoplastic. It is well known that the inferior olive is projected onto the cerebellar hemispheres, and the results of the studies of other authors indicate that the oral pole is projected onto the lateral portions of the anterior lobes. As has been mentioned already, Koster considers these portions to be new structures which would account for the defect in the oral pole of the olives. Furthermore, a restudy of the case on which Brouwer formulated his opinion shows that the lateral part of the anterior lobes was degenerated, which would account for the hypoplasia of the oral part only of the olives.

Koster's cases substantiate the theory of Masuda that the frontal part of the pons is connected with the caudal part of the cerebellum. Also the fact that there is no connection between the frontopontile and temporopontile tracts and the caudal part of the pons confirms Kapper's opinion that the latter is the oldest portion phylogenetically.

PEARSON, Philadelphia.

PSEUDOSCLEROSIS, WILSON'S DISEASE AND ENCEPHALITIS CHRONICA DISSEMINATA (WESTPHAL, STRÜMPELL, WILSON). W. SELETZKY, Arch. f. Psychiat. u. Neurol. **77**:704 (Aug.) 1926.

The symptom complex of pseudosclerosis was first described by C. Westphal, but it did not attract much attention until Strümpell advanced a more systematic

description of the disease entity on the basis of two cases of his own and on those of Westphal. According to him, pseudosclerosis sets in with purely motor symptoms in the form of general paralysis followed by spasticities, ataxia and tremors. These may occur in apoplectiform attacks and may show remissions. The tremors tend to be of the intention type; the voluntary movements are retarded; there are disturbances of speech in the form of slowness, monotony, indistinctness, and, at times, of the staccato type; the facial expression becomes somewhat fixed with a tendency to grimacing; there is no nystagmus; the gait is of the paretic-ataxic type; the deep reflexes are exaggerated, but the abdominal and cremasteric reflexes usually remain normal; there are no disturbances of objective sensibility, but there may be subjective ones in the form of pains in the arms and legs; control of the bladder and rectum is not affected. At times epileptiform attacks are noticed, especially toward the end. Psychically, there is generally a gradually progressing deterioration, at times acute excitements and hallucinatory periods, at others apathetic states. Impulsive emotional disturbances may be noticed. The course of the disease is chronic with remissions, but gradually progressive. It begins in the first or second decade of life without any discernible etiologic factors or hereditary influences. Postmortem examination fails to reveal any positive lesions.

This description by Strümpell dates back to 1898, and with a few exceptions was the generally accepted concept until 1912, when Hosslins and Alzheimer described definite microscopic changes in the disease. There were general destruction of nerve tissue, development of giant neuroglia cells that tended toward loss of nuclear chromatin and subsequent disintegration and secondary degeneration of the pyramidal tracts. Alzheimer thought that the characteristic feature of the disease histologically was the neuroglial change in the form of these giant cells with rich fibril formation, and that one was dealing here with hereditary predispositions.

With this clinicopathologic picture of Strümpell-Alzheimer as a central point the concept of the disease began to broaden out and to include a great many allied types. The syndrome was revised, and two new symptoms were added: (*a*) cirrhosis of the liver and (*b*) ringlike opacities of the cornea. About the same time Wilson reported a series of cases under the name of chronic lenticular degeneration, which he thought was more or less closely related to pseudosclerosis. The pathologica changes in this disease were, according to him, most pronounced in the putamen and pallidum, with secondary changes in other parts of the brain. The picture was complicated by cirrhosis of the liver and enlargement of the spleen. Clinically he described the following syndrome: tremors, rigidity and hypertonicity with contractures, dysarthria or anarthria, dysphagia and tendency to unmotivated impulsive laughter; psychically, a peculiar type of deterioration with a gradual constriction of the field of consciousness. There were no symptoms of the pyramidal tract. The onset is at an early age and progresses either rapidly (several months) or slowly (up to 7 years). It occurs usually in several members of the same family.

Ever since then, and up to the present time, the boundaries of these two disease entities have tended to overlap one another and to broaden out to include clinical and pathologic pictures belonging to other fields. In connection with extremes in this respect, such cases as were reported by Economo-Schilder, by the author, and Thomas, as well as a great many postencephalitic states, the author makes an attempt to decide whether such entities as pseudosclerosis on the one side, and Wilson's disease on the other really existed, and if so, what their respective syndromes were. Is it possible to establish a differential diagnosis between pseudo-

sclerosis and multiple sclerosis? Clinically, it seems to depend on four factors: (1) the early onset in pseudosclerosis; (2) the absence of known etiologic factors; (3) the presence of abdominal and cremaster reflexes; (4) the peculiar postmortem conditions. The first two, according to the author, do not differentiate the two disease entities because they are characteristic of both. As to the superficial reflexes, they are not invariably present in pseudosclerosis, and similarly not invariably absent in multiple sclerosis. As to the pathologic conditions, Spielmeyer has recently shown conclusively that the symptoms which were considered typical of pseudosclerosis are just as frequently found in Wilson's disease, and he considers the microscopic changes of the diseases essentially the same.

Recent studies of postmortem conditions in multiple sclerosis have shown that the plaques that were considered so characteristic of multiple sclerosis were found in cases of other parasitic diseases such as malaria, whereas cases of disseminated sclerosis at times show the absence of these plaques. This would point to the possibility that the existence of plaques is not the most important feature in multiple sclerosis, and that their absence does not justify the establishment of a new disease entity. Recent investigations of cases of Wilson's disease have shown that the original idea that the clinical picture was dependent solely on lenticular degeneration is not correct. Even Wilson mentioned the presence of lesions in other parts of the brain, but he thought that these have no bearing on the clinical aspect of the disease. Recently, however, a great many changes have been described in the cerebral cortex, in the caudate nucleus, Ammon's horn, substantia nigra and red nucleus. Spielmeyer is of the opinion that Wilson's disease cannot be regarded as a system disease, but as one with diffuse changes in different parts of the central nervous system. On the other hand, cases of chronic disseminated encephalitis were reported with clinical pictures typical of Wilson's disease.

All these considerations lead the author to the following conclusions: (1) Pseudosclerosis is not a special disease entity. Some of the cases are multiple sclerosis and others Wilson's disease. (2) Wilson's disease is really an encephalitis chronica disseminata.

MALAMUD, Foxborough, Mass.

THE ANATOMY AND CLINICAL ASPECTS OF PURE WORD DEAFNESS. PAUL SCHUSTER and HANS TATERKA, Ztschr. f. d. ges. Neurol. u. Psychiat. **105:** 494 (Nov.) 1926.

There is not any uniform conception of the anatomic substratum of pure word deafness. The Lichtheim-Wernicke concept of a subcortical sensory aphasia is not concurred in by many authors. Dejerine, Sérieux, Veraguth, A. Pick, Strohmayer and Quesnel consider the idea of a subcortical aphasia unproved, and look on an exclusive involvement of the cortex as essential for the production of the picture. Other authors, Blosen for example, consider the cortical and subcortical forms of word deafness as differences in degree of the same disorder. Liepmann is the only one who has found a pure subcortical lesion in a case of word deafness, but his patient developed a hemorrhage in this area shortly before death. Only three observers have reported left-sided foci, not always subcortical, in cases of word deafness; these are the cases of Liepmann, van Gehuchten and Goris, and Bloch and Bielschowsky.

Schuster and Taterka report the case of a woman, aged 67, who, in November, 1919, suffered an apoplectic attack with a resulting right hemiparesis which persisted throughout the illness. She apparently did not develop any immediate disturbance of speech. In 1921, about eighteen months after the onset, she developed loss of understanding for words, noises and melodies

which persisted until death two and one-quarter years later. Hearing was not affected — she showed absolutely normal hearing tests. With isolated exceptions she did not understand what was said to her. Her answers to questions were entirely irrelevant. When asked how she had slept, she replied that her name was Schramm. This was typical of her reactions. In repeating words after the examiner, it was found that if she understood the word she repeated it correctly, otherwise she replied wrongly; she could not repeat vowels. She understood certain words, particularly words with some emotional value to them. Spontaneous speech was good. She could give a good account of her illness. At times, but not often, she showed "verbal paraphasias," especially when she became excited. Thus she said "sissen" for "essen" at one time, "Taschenlappe" for "Taschenlampe," etc. She read without fault, and understood what she read. She wrote spontaneously with both hands and without error. Her understanding of melodies was disturbed. Simple tunes, either sung or played, were not recognized. She could not sing simple tunes, though it was determined that she knew and recognized the tunes which she was asked to repeat. Noises such as the rattling of keys, hand clapping and whistling were not recognized when the eyes were closed. Similarly the ticking of a clock, the clanging of knives, etc. were not recognized.

Necropsy showed definite changes in the brain. There was a left-sided area of softening which lay along the lateral ventricle, destroying the putamen and internal capsule. The paraventricular softening began in the frontal area and extended backward to the occipital area, forming a small, linear focus as it extended backward. The softening in the left temporal area involved Heschl's convolution and the white matter of the first and second temporal convolutions in their posterior thirds. In the right hemisphere there was a small cyst in the white matter of the anterior central convolution and a small focus in the right internal capsule. There were also pin-point foci in the putamen and thalamus on the left. The medial geniculate body on the left was atrophied.

Schuster says that the paraventricular focus was unquestionably the oldest, that it was the cause of the apoplectic attack, and that it had nothing to do with the word deafness since this is known to be caused by temporal lesions. Lesions in this area are common, are not associated with disturbance of speech and are nowhere near the speech area. He minimizes the significance of the areas of softening in the right hemisphere and believes they occurred shortly before death.

The aphasia, i. e., the word deafness, was due to the lesion in Heschl's convolution and to the destruction of the posterior third of the first and second temporal convolutions. Schuster believes his case is exceptional because of the lack of multiple foci. The foci in the left temporal lobe not only destroyed fibers as detailed, but also fibers connecting the temporal lobe with the operculum and the frontal lobe. The case substantiates the view of Lichtheim-Wernicke. Moreover, the cortex is not involved in the lesion, despite the contention of Monakow that the cortex must be involved in all such cases. "It has therefore been shown that the symptom of pure word deafness can be produced by a focus in the left temporal lobe." Sections of the case were sent to Mingazzini who did not find any evidence of involvement of the callosal fibers. Granting this, however, how can one explain word-deafness on the basis of a lesion of the temporal lobe? Various theories have been put forth. Some, such as Bastian, Dejerine, Pick and others, believe both temporal lobes must be injured, and Dejerine believes there is diminution in function of both auditory centers. Pick asserted that word deafness followed a bilateral lesion causing central deafness. Bastian believed there was a lesion of Wernicke's

area. Strohmayer and Veraguth attributed the word deafness to an incomplete bilateral lesion of the auditory field. Certain authors believe a lesion of the cortex is necessary; among these is Monakow. Schuster offers several hypotheses to explain the word deafness in his case: 1. Deep association fibers between Heschl's and Wernicke's areas may have been destroyed and in this way it was impossible to bring about a change from unqualified to qualified hearing. 2. This change may have been impossible because the projection fibers to Heschl's convolution may have been destroyed and hence the normal excitability of Heschl's area was not present. 3. It may be a combined effect of the first and second hypotheses. 4. Association fibers to the so-called area of Broca through the capsula externa and extrema may have been involved.

ALPERS, Philadelphia.

TUMORS OF THE CORPUS CALLOSUM. O. BALDUZZI, Arch. f. Psychiat. u. Nervenk. **79**:1 (Dec.) 1926.

An exhaustive clinicopathologic study of three cases of his own and a review of 113 cases in the literature are used as a basis of a critical analysis of the symptomatology, localization and differential diagnosis of tumors of the corpus callosum. The symptoms found in this material are summed up under three headings: 1. General Symptoms. (*a*) Symptoms of the optic disk occur in different forms from ordinary neuritis to definite choking. The frequency of definite symptoms is about the same as in a number of other types of tumor of the brain, and could not be used for differential diagnosis. (*b*) Headache is somewhat less frequent than in other tumors; it comes on early in the disease, and progresses with the psychic symptoms. (*c*) Vomiting is even rarer than headache. (*d*) Generalized epileptiform convulsions are not frequent enough to serve as a symptom of this type of tumor, yet too frequent to be of help when absent. (*e*) Vertigo is rare and is of the subjective type.

2. Remote Symptoms: Under this heading the author discusses involvements of the cranial nerves and comes to the conclusion that they occur rarely, mostly in tumors that involve the anterior portion of the corpus callosum and affect the frontal lobe. The facial nerve is most frequently affected, and the damage is of central type.

3. Symptoms Due to Direct Effects of the Tumor: (*a*) Disturbances of motility are common and consist of bilateral and monolateral disturbances of motility, usually in the form of paresis or paralysis. In some instances, however, one finds at certain stages signs of irritation only in the form of reflex disturbances. Disturbances of bilateral motility are found by far most frequently in tumors of this localization. (*b*) Objective sensibility disturbances are rare and are most frequent in tumors of the posterior part of the corpus callosum which are liable to affect the thalamus; they involve both superficial and deep sensibility, although in some rare instances only superficial sensibility was affected. Frequently one finds station and coordination disturbances, but the importance of this has been somewhat exaggerated by those who failed to recognize the possible dependence of such disturbances on general paralysis. Similar conclusions are reached also as regards ataxia. (*c*) Psychic disturbances of one type or another are frequent (absent only in ten of 116 cases). From the rich variety of possible psychic symptoms, Raymond has abstracted a syndrome which the author considers characteristic of tumors of the corpus callosum. It appears early and includes dissociation of ideas, bizarre behavior, disturbances of memory affecting recent occurrences mostly, a marked character change, and emotional instability. As a localizing sign this syndrome must occur at the beginning of

the disease, without other psychic components and without somatic disturbances. (*d*) Apraxia of the face, tongue and extremities, especially of the left side, is one of the most useful signs for localization of the tumor in the corpus callosum, although the absence of this symptom does not exclude a tumor there. (*e*) Disturbances of speech are rare, but are nevertheless observed. They may be of aphasic or of dysarthric type.

Following this analysis the author comes to the conclusion that the following symptoms may be regarded as indicative of corpus callosum localization: (1) apraxia of the face, tongue and extremities (left or sometimes bilateral); (2) bilateral motility disturbances; (3) the psychic syndrome described by Raymond; (4) no involvements of the cranial nerves.

The localization of the tumor in any particular segment of the corpus callosum is much more difficult and one can speak of probabilities only. Involvement of the cranial nerves, especially of the second and third nerves, would speak for involvement of the anterior segment. Definite hemiplegia or disturbances of sensation point to the middle and posterior segment, whereas involvement of the optic tracts with hemianopia would point to the splenium.

The diagnosis of a tumor in the corpus callosum is best reached by the process of elimination. Of other tumors, those that come up most frequently in diagnosis are located in the frontal, parietal or temporal lobes. The presence of apraxia of the type described, bilateral motility disturbances, psychic disturbances without involvement of the cranial nerves (especially the third nerve) help in differentiation from a tumor of the frontal lobe. The type of psychic disturbances and their relation to the speech or sensory disturbances help in differentiation from tumor of the temporal lobe. When the syndrome of the corpus callosum outlined is not clearly evident, sensory aphasia may render differential diagnosis difficult. In such cases, if the clinical picture suggests left-sided localization, sensory aphasic disturbances will determine the diagnosis. The differential diagnosis is still further complicated by the fact that tumors of one of the temporal lobes, if extensive enough, may involve the peduncle of the opposite side. In these cases, however, there should be distinct signs in the cranial nerves which would speak against a lesion of the corpus callosum. The presence of superficial along with deep sensibility disturbances, or of superficial disturbances only, with the typical psychic syndrome and apraxia, will help to differentiate tumors of the corpus callosum from those of the parietal lobe. Sometimes the question of differentiating a lesion of the corpus callosum from general paralysis comes up, but there, of course, the serologic symptoms will be most useful. A review of the pathologic conditions shows that these tumors are most frequently gliomas. Next to these are sarcomas, and then gliosarcomas. Lipoma, gumma, myxosarcoma and cancer are found much less frequently. The growth of a tumor primarily originating in the corpus callosum practically never follows the callosal radiations. Therapeutically, surgical intervention is hardly to be considered, and the author thinks that deep roentgen-ray treatment is probably the most practical, although the prognosis in any case is poor.

MALAMUD, Foxborough, Mass.

PATHOLOGIC-ANATOMIC INVESTIGATIONS ON THE RELATION OF HUMAN ENCEPHALITIS TO HERPES, KLING, AND DISTEMPER ENCEPHALITIS. HANS-PETER KUTTNER, Ztschr. f. d. ges. Neurol. u. Psychiat. **105**:182 (Oct.) 1926.

The question of the relation of epidemic encephalitis to other inflammatory diseases of the nervous system and to experimental forms of encephalitis is still unsettled. Attempts to reproduce epidemic encephalitic in animals go

back to Economo and Wiesner. *Diplostreptococcus pleomorphus,* attributed by these authors to be the cause of encephalitis, causes lesions in animals in no way similar to those found in man. Bradford, Wilson and Levaditi thought the cause was a filtrable virus, such as the virus of herpes which causes lesions similar to those in epidemic encephalitis. Many viruses have been reported as the cause of encephalitis which have later been found to be unspecific. Among them are: the Thalhimer virus, the Kling virus, a virus related to rabies and numerous others. The filtrable viruses of the herpetic type, which Levaditi and others gathered into a group and found to be related to smallpox, rabies and poliomyelitis, appear clinically, pathologically and epidemiologically to be similar to epidemic encephalitis in man. In experiments on animals, Levaditi has established a crossed immunity between the herpetic and the encephalitic virus. Many investigations have been carried out in the attempt to establish the relationship of herpes to encephalitis. The most recent of these is that of Flexner, which like the others did not establish a relationship between the two diseases. Kuttner remarks that the only positive result of the many researches has been the establishment of the fact that the true virus of epidemic encephalitis has not yet been discovered.

Kuttner studied the pathologic picture in human encephalitis and in herpes, Kling and distemper encephalitis to determine whether there was any relation between the encephalitis of man and that of animals. Direct transmission had failed, so he studied the pathologic picture in these various types. In the human type "there is no doubt today that in almost all the cases the inflammatory changes are limited to the brain stem and the central ganglia, less often affect the cord and still less often the cerebrum and cerebellum." The severest damage is in the subthalamic region, pons and medulla, especially around the aqueduct and fourth ventricle. The greatest changes occur in the substantia nigra. "The occurrence of infiltrative and degenerative changes in the substantia nigra in human encephalitis is on the whole constant." This differentiates epidemic encephalitis from other forms of encephalitis. According to Trétiakoff and Marburg, the changes in this region are present in the earliest stages of acute epidemic encephalitis.

In contrast to the picture in human epidemic encephalitis, Steiner found that herpes encephalitis causes the most marked damage in the basilar portions of the cortex. There is a meningo-encephalitis of the cortex with relatively slight involvement of the basilar portion of the brain, with an outspoken tendency to necrosis, and a clear tendency to a layer-like arrangement of the changes. Others have reported purpuric foci in the brain.

Kling encephalitis (*Encephalitozoon cuniculi*) affects chiefly the kidneys and liver. Severe pathologic changes in the nervous system are lacking. Slight infiltrations of lymphocytes and plasma cells are found localized in the cortex and in the midbrain.

In distemper encephalitis there is a predominant involvement of the plexuses, ependyma and adjacent parts, and of the outer surfaces of the brain stem, cerebrum and cerebellum. The pathologic conditions are not specific.

Kuttner examined twelve cases serially: three rabbits had herpes encephalitis, three rabbits and a dog had the Kling virus, and the rest had distemper. Those infected with the herpetic virus did not show involvement of the substantia nigra. One animal infected with the Kling virus showed a slight involvement of the substantia nigra. The other two and the animals with distemper did not show involvement. In other words, the substantia nigra was not involved in any of the cases of animal encephalitis.

ALPERS, Philadelphia.

CLINICAL AND HEREDITARY FEATURES OF DEGENERATION PSYCHOSES. K. KOLLE, Arch. f. Psychiat. u. Nervenk. **78**:731 (Nov.) 1926.

This article reports the results of an investigation of the clinical features and transmission of the so-called degeneration psychoses in a clan comprising 141 members and spreading over four generations. The starting point was furnished by two members of this clan who were studied by the author and who presented similar recurrent psychotic episodes of a rather unique type. These psychoses, although showing a tendency to recur, were not otherwise typical of the manic-depressive syndrome; at the same time, they were not characteristic of schizophrenias or other previously established syndromes. In both cases the attacks were at times precipitated by somatic diseases (diphtheria and other conditions); at others, as reactions to difficult environmental situations, and at still others spontaneously. The character of the disturbances in the two patients, as well as during the different attacks, had the following points in common: they tended to come on with acute confusion, disorientation, misinterpretations, and transient hallucinatory periods; there was a tendency toward mood depression, even during the psychomotor activity; there was a strong erotic element, and after a short or longer duration, the attack passed without leaving any apparent intellectual or emotional defect. The detailed analysis of these two cases (one of which was classified as an atypical manic-depressive psychosis, and the other left without classification but described as a recurrent compulsion psychosis with depressive phases) led the author to regard them as belonging to the reaction type described especially by Kleist under the group of degeneration psychoses. Since, in this group, hereditary influences seem to play a particularly important rôle, the present investigation was undertaken.

Among the 141 members, the author found at least seven definite psychoses with a number of psychopathic personalities. The psychoses were all of a type that, just like the two described, could not be made to fit into any of the previously established classification groups and could be ranged with Kleist's constitution types only. This large group of mental disturbances that Kleist regarded as falling into the constitutional degeneration types were considered by him as having a strong leaning toward homonomous (syntonic) form, but showing so many characteristics foreign to the manic-depressive psychoses and such a tendency toward occurrence in the same familial groups that the establishment of a new syndrome was considered justified. In different persons and during the different attacks, all of the three subgroups described by Kleist were found: autochthonous lability (attacks occurring on a presumably spontaneous basis), reactive lability, and symptomatic lability. The author agrees with Kleist and Binswanger that here one is dealing with phenomena that are fundamentally dependent on a constitutional degenerative predisposition. Those members of the clan who showed no definite psychoses, but were distinctly psychopathic personalities, showed leanings toward the same types of reactions as were shown by the psychotic members, although not to the same extent. It appears that although the members of the fourth (to which the patients reported by the author belong) and the third generations showed all of the three types mentioned, the first and second showed more distinct leanings toward either the one or the other, and so one can see a leaning toward the reactively labile constitution on the maternal side of the clan, and toward autochthonous lability on the paternal side.

The attempt to discover other constitutionally transmitted factors, either of a bodily or mental type, was not successful. A tendency toward hereditary transmission of a pyknotic disposition (Gruhle) is at best a probability. In

the other types described by Kretschmer, the author could not find such tendencies, and there was no particular relation between the psychopathic or psychotic reaction types and those of bodily development.

In an appendix to the article the author reports the results of a similar but uncompleted study of another clan. The main trends shown are similar to those in the first clan, but in this case there was a tendency to the heteronomous rather than to the homonomous type of reaction, so that the psychoses have more leaning toward the schizophrenic type. The author suggests that further study be undertaken: (1) to establish this psychopathologic reaction type as a definite clinical entity and (2) to investigate the advisability of further subdividing the group into homonomous and heteronomous types.

MALAMUD, Foxborough, Mass.

EARLY EMBRYONIC CAUSES OF MALFORMATIONS OF THE NERVOUS SYSTEM. ERNST DE VRIES, Schweiz. Arch. f. Neurol. u. Psychiat. **19**:274, 1926.

Earlier investigations have taught us a great deal concerning the causes of malformations of the central nervous system. Much, however, remains unknown. The attempt to attribute status verrucosis deformis (Ranke) of the cerebrum and certain heterotaxias of the cerebellum to pathologic processes of the membranes seems justified.

De Vries reported a number of cases. The first was that of a human fetus of 9 months, with polygyria of the cerebellum. Important observations were that the polygyria involved the three outer layers only, and that the leptomeninges in the sulci of the diseased areas were absent. As a result, the adjacent structures became fused. De Vries believes that an early inflammation of the membranes took place; this left a thickened pia, as a result of which this membrane could not keep pace with the formation of the convolutions, which, in turn, resulted in adhesions between the convolutions and prevented the formation of normal secondary convolutions. Over the occipital lobe there was a definite meningitis with thickening and infiltration of the leptomeninges. The underlying structures were involved secondarily, probably due to ischemia.

Case 2 was one of heterotopia and heterotaxia of the cerebellum. The patient was a simple-minded girl who died at the age of 17 years. There had been prominent cerebellar symptoms but no history of previous brain disease. Anatomically there was slight microcephaly, simple type of cerebral convolutions, marked atrophy or agenesis of the cerebellum and doubling of the granular layer. In the vermis there were two symmetrically placed nodules of gray matter the size of a pea. Whether the picture produced was traumatic or meningitic, could not be determined.

Case 3 was one of status verrucosis deformis of the cerebrum and extensive heterotaxia of the cerebellum. The child had been an idiot and had died at the age of 9 years. As an infant, he had apparently been well, and only gradually did the parents become aware of the mental deficiency. There had been slight convulsions, and he had had an attack of inflammation of the lungs. His movements had been extremely spastic and ataxic, the extremities had become spastic, and the posture had been one of extreme opisthotonos. The cerebellum showed a large number of heterotactic areas. Investigation of these revealed the interesting fact that there was no pia between the convolutions, and that these were intimately fused. In this vicinity, there was no evidence of an early meningitis. Examination of the remaining structures showed some scar formation in the cerebellum, heterotopia of the Purkinje cells, thickening of the pia covering the cerebrum with marked clouding at the tips of the temporal lobes,

enlargement of the ventricles, microgyria and polygyria of the cerebrum, the so-called status verrucosis deformis, and an excessive folding in the region of the external geniculate body.

The fourth case was one of status verrucosis deformis of one hemisphere. The patient had been epileptic and feebleminded since childhood. There was no history of encephalitis. There had been a spastic infantile hemiplegia on the right side. He died at the age of 54 of tuberculosis, without evidence of meningitis. At necropsy there was an accumulation of pus over both hemispheres. The convexity of the left hemisphere showed a typical microgyria. This whole hemisphere was small and the opercular formation was only indicated. In the region of the microgyria, the three outer layers showed excessive folding, whereas the two deeper layers showed the normal convolutional structure. The pia was seen to extend into the principal sulci; however, it did not extend into the deformed sulci.

It would seem from these observations that the formation of the gyri and sulci can not progress far when the pia fails to follow. In contradistinction to Brun, who attributed many forms of malformation to a toxic endogenous cause, De Vries believes that at least one type may be explained on a mechanical basis and is a result of disease of the meninges.

WOLTMAN, Rochester, Minn.

THE INDICATIONS AND RESULTS OF SECTION OF THE CERVICAL ROOTS IN THE SURGERY OF PAIN. R. LERICHE, J. de neurol. et psychiat. **27**:17 (Jan.) 1927.

Leriche reports his results with section of the cervical roots in the treatment of painful syndromes. He has done thirty-two ramisections in all, in twenty-eight of which there had been pains in the limbs. Of these twenty-eight, sixteen were in the cervical region; it is these sixteen cases which he reports.

The cervical communicating roots are of two types — superficial and deep. The superficial roots take origin from the superior cervical ganglion and especially from the stellate ganglion. They come in variable numbers from the stellate, from four to seven or eight, and pass to the anterior branches of the cervical nerves and the anterior root of the first dorsal nerve. The deep fibers take origin from the vertebral nerve and cannot be reached easily, whereas the superficial roots can. Physiologic experiments on these cervical roots have given important results. Electrical stimulation of the roots has caused painful effects in various areas which are variable for each root, and also has produced hypotension. Electrical stimulation of the second and third cervical produced: (1) pain in the external ear and in the teeth of the lower jaw, and (2) rhythmic movements of contraction and dilatation of the pupil with alternating propulsion and retropulsion of the eye. Electrical stimulation of the seventh cervical produced lively pain in the inner aspect of the arm and in the hand, however, without a radicular or truncular type of distribution. Stimulation of the upper pole of the stellate ganglion caused pain in the arm and in the precordial region.

Section of the roots caused painful phenomena, muscular phenomena, vasomotor, ocular and pharyngolaryngeal symptoms. The muscular and sensory effects were inconstant. In only two cases did pain occur in areas similar to those involved by electrical stimulation, viz.: in the jaw and lower teeth. Two patients complained of frontal and occipital headaches, which were transitory however. In one case the headache lasted several months. It was more constant if the ganglion was ablated. Cutaneous sensibility always remained intact after ramisection; muscular power was not diminished, and there was no atrophy. In one case there was atrophy of the pectoralis major, the shoulder muscles and the interossei. This atrophy disappeared after one year and was

attributed to injury of the brachial plexus during operation. Vasomotor signs were constant after ramisection. Soon after operation there is hypertension both ipsilateral and contralateral. This phase of hypertension is brief but constant. Arterial pressure falls after three or four hours, and in from three to four days again reaches the preoperative level. The blood also shows changes after ramisection. There is an increase of red and white blood cells in the peripheral circulation. In one case there were 4,200,000 erythrocytes before operation, 6,380,000 after operation and 5,100,000 the next day. White blood cells rose from 5,200 to 19,300, all polymorphonuclear cells. Pharyngolaryngeal phenomena were observed also after section of the cervical roots. Patients complained of a full throat and some change in voice which was usually transitory. Objectively, the pharynx and larynx on the side of operation were hyperemic and edematous. Section of the first dorsal root always produced the Claude Bernard-Horner syndrome. The palpebral signs in such instances were always more pronounced than when the superior cervical ganglion was ablated.

Leriche has performed cervical ramisection in angina pectoris, Raynaud's disease, in diffuse sympathalgias, ascending neuritis, and in a case of syringomyelia. Leriche operated on four patients who had angina, which gave relief from pain. He advises against ablation of the superior ganglion, either unilateral or bilateral, because this has been shown to be dangerous experimentally. In one animal which came to autopsy after such an operation there was myocardial degeneration. He advises ramisection for this condition. He has obtained encouraging results in the treatment of Raynaud's disease by ramisection.

ALPERS, Philadelphia.

IODIZED OIL TEST IN TUMORS OF THE SPINAL CORD. B. BROUWER and I. OLJENICK, Acta psychiat. et neurol. **1**:15, 1926.

The authors report in detail the histories of two cases of tumor of the spinal cord, in which the diagnosis and localization of the lesion by clinical methods was uncertain. The first patient had complained of a pain in the back, buttocks and legs, of gradually increasing severity, for a period of six years. For a year and a half previous to admission to hospital, this pain had increased greatly, the legs had become weak, and the patient was constipated. On examination, a mild degree of wasting of the left leg was noted; there was weakness in flexing and in extending the knee on that side, and there were slight involuntary movements of both feet. She complained of subjective sensory disturbances below the second lumbar dermatome, but objectively there was hypesthesia only around the anus and on the dorsal surfaces of the legs. The sense of position was lost in the big toes. The gait was uncertain. The knee and ankle reflexes and the left plantar reflex were absent. The cerebrospinal fluid was under normal pressure, but it was yellow and the globulin reaction was strongly positive.

Although these symptoms were suggestive of tumor of the lowest part of the spinal cord, it was difficult to know whether the lesion involved the cauda equina only or both the cauda equina and the conus. Injection of iodized oil 40 per cent showed a block between the twelfth dorsal and first lumbar vertebrae. At operation over this point, a fibroma lying to the right of the spinal cord and pressing the cauda equina to the left was found. The tumor was removed successfully, and the patient made a good recovery.

The second patient had noticed a gradually increasing weakness of the legs and some involuntary jerkings. Examination showed both legs to be distinctly weak, with increased knee and ankle reflexes and patellar clonus. The reflexes

of Babinski, Mendel-Bechterew, Chaddock and Oppenheim were present on both sides. The sensations of touch, pain, heat and cold were diminished over the entire right leg. The sense of position was lost in both big toes. The gait was spastic and the tonus of the muscles of both legs was increased. The cerebrospinal fluid was under normal pressure, and the test for globulin was slightly positive.

During the next five months, the condition progressed until the symptoms were those of a transverse lesion of the spinal cord. Certain additional psychogenic symptoms, however, still made the diagnosis uncertain. Injection of iodized oil by the cisternal and lumbar routes showed an obstruction opposite the body of the tenth dorsal vertebra. Operation at this point revealed an endothelioma psammosum, which was removed completely. The patient made a good recovery.

These two cases, as well as three others reported by the authors in a previous article, lead them to conclude that the diagnosis of tumor of the spinal cord is uncertain unless confirmed by the injection of iodized oil. So strongly do they feel the usefulness of this method, that they divide the history of the diagnosis and treatment of tumors of the spinal cord into the preiodized oil and the iodized oil epochs. They have never observed any disagreeable consequences from its use, and they point out that it may be used as freely in locating a lesion of the upper portions of the spinal cord as of the lower. The use of ascending iodized oil is of value in localizing the lower level of the tumor and thus limiting the length of the operative incision, with a consequent facilitation of recovery.

PEARSON, Philadelphia.

A FORMULA FOR THE STAINING OF AMEDULLATED FIBERS, AND CENTRAL AND PERIPHERAL NERVE ENDINGS IN SECTIONS. S. R. CAJAL, Trav. du lab. de recherches biol. de l'univ. de Madrid **23**:237 (Nov.) 1925.

Pyridine-Silver Nitrate and Alcohol

1. Frozen sections (from 30 to 40 microns) of material preserved in formaldehyde (for three or more days) are quickly washed in distilled water and immersed for from four to six hours in the following solution:

2 per cent aqueous solution of silver nitrate	10 cc.
97 per cent alcohol	5 to 6 cc.
Pure pyridine	7 to 10 drops

At the end of four hours, sections should appear light brown. If they are too light, place a Stender dish with the sections over an alcohol lamp for a few minutes. A curved glass rod should be used to transfer sections to the different solutions.

2. A few sections (three or four) are transferred to a small Stender dish containing 8 or 10 cc. of absolute or almost absolute alcohol (from three to six seconds).

3. From the alcohol, sections are put in the following reducing mixture:

Hydrochinone	0.30 Gm.
Distilled water	70 cc.
Formaldehyde (40 per cent)	20 cc.
Acetone	15 cc.

In this mixture, they acquire a yellowish brown or deep brown color (from one to three minutes).

4. Wash thoroughly in several changes of water, tone with yellow gold chloride, fix with sodium thiosulphate.

5. Wash again in water; spread the sections on a slide, mop with filter paper and dehydrate with absolute alcohol, followed by oil of clove, xylene and dammar.

The method just described is constant in its results and stains, with great sharpness, the baskets, the mossy and climbing fibers of the cerebellum, the calixes of Held of the nucleus of the trapezoid body, the nerve endings of the ventral ganglion of the acoustic nerve, etc., and in addition, the motor plates, sensory nerve endings and diverse details of muscular, nervous and epithelial tissues.

Too much extraction of silver nitrate when the sections are in the absolute alcohol (2) results in light staining; this defect can be overcome through the addition of three or four drops of 2 per cent silver nitrate solution to the reducing mixture (3). If the white matter fails to stain well, place the sections, before immersion in the silver nitrate solution, in the following mixture (from two to four hours):

Water	10 cc.
Alcohol	10 cc.
Ammonia	10 drops

Wash in water and transfer to silver solution (1).

A second formula, simpler but not so constant is given. Frozen sections of material that has been fixed in formaldehyde are immersed in the following combined staining and reducing mixture:

2 per cent solution of silver nitrate	10 cc.
Pyridine	10 drops
Formaldehyde, 40 per cent (not neutral)	7 to 10 drops

until they acquire a deep brown color (from four to six hours). Wash in three changes of water, tone with gold chloride, etc. The best results were obtained in the cat.

NONIDEZ, New York.

COMPLETE SPASMODIC MONOPLEGIA OF THE LEFT FOOT OF CORTICAL ORIGIN. VIGGO CHRISTIANSEN, Acta psychiat et neurol. **1**:39, 1926.

Christiansen calls attention to the number of instances in which cases of epidemic encephalitis are not recognized as such and in which cases of other neurologic conditions are diagnosed as encephalitis. The case he reports illustrates the latter occurrence. A woman, aged 37, was healthy until 1918, when she suffered from an uncomplicated attack of influenza, which lasted five days. Following this illness, she suffered from severe headaches. Three months later, she had a convulsion on the street, and these attacks have occurred at frequent intervals since that time. The seizures commence in the left toe, spread upward along the limb, involve the thoracic musculature on the left side and terminate in the left arm. Sometimes she loses consciousness. Following the first few attacks she noticed that the left leg would be weak. Gradually this weakness became more prolonged until the leg was paralyzed. Five days before admission to the hospital, the left arm became paralyzed with almost apoplectiform suddenness. This paralysis lasted two or three hours. For the last seven years, she had suffered from severe headaches with exacerbations and attacks of vertigo. Recently memory had become defective. She was referred with a diagnosis of postencephalitic syndrome.

On examination she was found to have a complete spastic paralysis of the flexors and extensors of the left foot, with increased reflexes and a Babinski sign. There was weakness of the left arm, and it was suspected that the lower part of the face on the left side was weak also. At the time of this examination, there was weakness of the muscles of the anterior part of the right leg and a Babinski sign on that side. She was exhausted by a long journey, and the last observation was not made subsequently.

On the basis of the distribution of the paresis on the left side and the fleeting paresis of the right leg, the author diagnosed a tumor of the right cerebral hemisphere adjacent to the median line. A tumor in this position would press also on the left hemisphere, inhibiting its vicarious control of the left leg and making the paralysis in that extremity complete.

Operation revealed an extracerebral tumor mostly on the right side, but pressing over to the left. It was removed completely. Following the operation, the left hemiplegia became complete. (The author states that such a result always follows the extirpation of extracortical tumors and is due to the sudden relief of pressure by its removal.) More interesting still, the right foot became paralyzed completely, demonstrating that the observations on admission indicated the menace to the left hemisphere. After a short time, all these symptoms disappeared, leaving only a spastic monoparesis of the left foot, which was most evident in the extensors. This is improving also. In this connection it must not be forgotten that the tumor was present for seven years, and that there must be degeneration in the pyramidal tract fibers from the centers for the feet in the right hemisphere.

The transitory paralysis of the left arm was a puzzling symptom. Such a condition is due usually to multiple sclerosis or syphilis. It is rare in tumor, and when it does occur the paralysis usually persists, because it is produced by hemorrhage into the tumor. In the case reported, it may have resulted from some alteration of the vascular and cerebrospinal fluid circulation.

Cases such as this are not rare, but are diagnosed inaccurately in most instances, because the examiner neglects to place proper emphasis on such symptoms as the spastic paralysis and the weakness in the left arm and the lower part of the left side of the face.

PEARSON, Philadelphia.

DISTURBANCE OF VIBRATORY SENSATION AS A SYMPTOM OF MULTIPLE SCLEROSIS. LUDWIG MANN, Ztschr. f. d. ges. Neurol. u. Psychiat. **104**:443 (Sept.) 1926.

Disturbances in sensation play a relatively small part in the symptomatology of multiple sclerosis. Mann has observed disturbance of the vibratory sense so constantly and for such a long time in multiple sclerosis that he considers it a symptom pathognomonic of the condition. Claude and Egger (1906) found the vibratory sense impaired in twenty of twenty-two cases of multiple sclerosis, either in the form of increased or decreased sensation. No other satisfactory studies of vibration in multiple sclerosis have been made, or if vibrations have been observed, they have been accompanied by other sensory disturbances. The observations of Mann were made independently of those of Claude and Egger and are entirely in accord with them.

Mann observed outspoken disturbances in sensation in thirty-two of thirty-seven cases of multiple sclerosis. Of the other five patients, three were markedly improved, and in the other two cases the diagnosis was doubtful. He used a tuning fork with 128 vibrations to the second, testing four points — the internal malleolus of the foot, the patella, a malleolus of the hand (the radial usually) and the olecranon. The tuning fork was struck and set in vibration, it was

placed on one of the parts mentioned, and the patient was asked if he felt anything. Tested in this way, it was found that disturbance in vibration was most marked in the feet, less in the patella, and least in the upper extremities. In seventeen cases a vibratory disturbance was found only in the feet, in ten cases also in the patella, and in five cases in the upper extremities also, in addition to the other two places. Disturbance in vibration is never present in the upper extremities when it is normal in the feet. While vibration was disturbed in these cases, the other types of sensation were normal. In fifteen cases there were no other sensory disturbances, while in seventeen cases slight disturbance of the sense of position in the big toe was found. "This isolated occurrence of disturbance of vibratory sense with the lack of other disturbances in sensibility appears to me to be particularly characteristic of multiple sclerosis, and is perhaps of as much value diagnostically as the loss of the abdominal reflexes."

The course of the vibratory fibers is still unsettled. Whether vibration is a particular type of sensation, or whether it is a part of the deep sensibility, whether its sensations come only from the bones and periosteum, or from all tissues, is undetermined. Frank has recently claimed that the fibers which carry sensation pass to the dorsal roots into the dorsal horns, from there to the anterior horns, and then backward to reach the lateral pyramidal tract. According to this, any disease affecting the anterior horns or the lateral tracts should give vibratory disturbances. Frank claims to have found this in poliomyelitis, amyotrophic lateral sclerosis, syphilitic spinal paralysis, multiple sclerosis and other conditions, and considers this disturbance in diseases of the pyramidal tract as more constant than the Babinski sign. Mann is unable to confirm these observations. He has never found disturbance in vibration in new or old cases of hemiplegia. The only disease in which he has found an isolated disturbance in vibratory sense without the involvement of other types of sensation is multiple sclerosis. He considers the disturbances pathognomonic for this disease.

ALPERS, Philadelphia.

THE MOTOR PLATES OF THE MAMMALIAN TONGUE. S. R. CAJAL, Trav. du lab. de recherches biol. de l'univ. de Madrid **23**:245 (Nov.) 1925.

The motor plates of the tongue of the rabbit are usually placed near the ends of the muscle fibers, in the vicinity of the fibrous septa in which the fibers are inserted. This arrangement, already noted by many investigators in certain muscles of fishes and amphibia, has also been reported by Tello in the developing motor plate. Each terminal branch encloses a bundle of neurofibrils, which in the varicose portions and terminal enlargements are arranged in networks of fibrils separated by abundant neuroplasm. The finest branches end in rings or in free loops.

Cajal failed to find Ruffini's ultraterminal fibers and Bremer's accessory fibers in the tongue of the adult rabbit, cat and young mouse. Thin fibers present in sections of the tongue of these mammals were collaterals of axons of nerve bundles in the vicinity. In some sections, however, there appeared fine branched fibers forming anastomoses between two nerve bundles.

Despite his failure to find accessory sympathetic fibers in his slides, Cajal does not deny their existence. However, he emphasizes the fact that histologic demonstration of such fibers is extremely difficult (it is hardly possible, in fact, to differentiate accurately a fine amedullated collateral arising at a long distance in a medullated fiber from a sympathetic fiber). Furthermore, he was impressed by the reflections and criticisms of such an authority on the sympathetic nervous system as Langley, but since the problem can only be solved

in an objective manner he preferred to withhold judgment until he has carried out more complete and extensive observations. A point that might have a bearing on the presence of accessory fibers is the frequent occurrence of "evolutive errors" in the development of both central and peripheral nerve endings, on which Cajal extensively commented in his contributions on the ganglia, spinal cord and medulla of the embryo, on the development of the retina and cerebellum and, finally, on the regeneration of the nerves. Tello, Castro and Vadillo also published observations on the same question. On the strength of these observations, Cajal ventures the opinion that the accessory fibers might represent a persistence of some of the abnormalities present during development that usually disappear in the adult on account of their physiologic uselessness but which may persist in a more or less degenerated form in some instances.

The intraplacular network of Boeke is discussed. This structure did not always appear stained in the motor plates, a fact previously reported by Boeke. Since the network could not be stained with methods that gave good results for the staining of the nerve endings, Tello suggested that the structure described by Boeke and his students was a product of coagulation. Cajal is inclined to accept the preexistence of the network but has not been able to find definite proof of its connection with the neurofibrils of the nerve endings. He believes that the network represents the framework of the plate and that it is continuous with the striated substance of the muscle fiber. Facts in support of this opinion are given and discussed, but the author warns that his interpretation is not final and that new studies might prove it to be incorrect.

The technic used for the preparation of the slides was the method previously described of staining with pyridine-silver nitrate and alcohol.

NONIDEZ, New York.

GRAPHIC REPRESENTATION OF PAST-POINTING, DEVIATION AND ORIENTATION, WITH SOME REMARKS CONCERNING THE CENTRAL MECHANISM. BÁRÁNY, Riv. oto.-neuro-oftal. **3**:551, 1926.

The registering of three different tests following vestibular stimulation has had a long history, one not altogether pleasant for the author. In several ways he sought to perfect this so that it might be registered, but he found that errors were inconstant and that subjective feelings became more and more prominent. Suggestion and deception throw the patient off and do not improve his answers.

These experiments led Bárány to the consideration of the errors from a theoretic standpoint. In order that a man may regain a certain point in space after once touching it, there should be a certain sensation in the skin of the finger, a certain sense of position in the shoulder and a certain sensation of movement. If the arm is 75 cm. long, it can make a circle of 471 cm. Deviation of 1 cm. on the paper corresponds to 45 minutes, 51.5 seconds, or about 0.75 degree. Sometimes the error may be from 2 to 3 cm., but the deep sensibility of the shoulder has a threshold of 0.2 to 0.4 degree, so that the cause of the error is not in the imperfect development of sensibility. Taking up a hollow sphere and noting positions from 0.1 to 0.2 degree from one another, one obtains a total of 810,000 positions, each separated from another by a space through which the movement at the shoulder is appreciated. Between two such positions, however, there would be a minimal difference, and it must be supposed that in the cortex these two positions would distinguish themeselves slightly. A great majority of nerve cells innervated in these two different positions

would be the same, there being only a difference in innervation of certain cells, some being stimulated more strongly by one position than by the other. Another hypothesis is that in the two positions the vast majority of cells would be equally stimulated, but that a few cells would be stimulated by position *A* that were not stimulated by position *B*. Such differentiation is nicely done in the exercise of taste and hearing. Now, in the parietal cortex, the lamina receiving impulses from the periphery contains 135 cells per 0.01 sq. mm. This layer is 0.3 mm. broad; therefore, there are in a column, with a surface of 0.1 by 0.1 mm. and a depth of 0.3 mm., 405 cells, and in a column represented by 1 sq. mm. surface, 40,000 cells. The cells which would be necessary to differentiate the 810,000 different positions would thus occupy a space of 20 sq. mm. It is known, however, that the territory presiding over the deep sensibility of the whole body must be about 4,000 sq. mm. Granting that there is a barely equal sensibility over the whole body and that the number of thresholds depends naturally on the size of the excursion of the limb, one arrives by an incomplete conclusion at the quantity of 400 sq. mm. It might be concluded from this that the threshold of sensibility has 10 cells at its disposition. Such reasoning might aid in calculating the size of such foci in the center for the different articulations, and after the same reasoning one might calculate similar figures for the optic, acoustic and motor mechanisms. Comparative anatomy may give additional information. This is merely a hypothesis, which facts and deeper reasoning may prove or disprove.

FREEMAN, Washington, D. C.

LIMITATIONS AND POSSIBILITIES OF ENCEPHALOGRAPHY. M. FISCHER, Arch. f. Psychiat. u. Nervenk. **79**:96 (Dec.) 1926.

Reporting the results of eighteen consecutive encephalographies, the author emphasizes that although encephalography has not fulfilled all expectations in diagnostic as well as therapeutic value, it has passed the stage where it can be considered a dangerous procedure. He dismisses the idea that in all cases without exception the direct ventricular method of Dandy should be undertaken. This method is the one of choice in cases of posterior fossa growths. All other conditions are just as safe with the Bingel method. The average quantity of fluid removed in his eighteen cases was 80 cc., and they were all done in the sitting position. The fluid is removed slowly, the patient having received no food for a considerable time, thus avoiding the danger of vomiting.

The eighteen cases fell into the following groups: (1) Epilepsy, six cases. The first two were clinically genuine epilepsy. In the first, encephalography showed marked atrophy of the cortex, and in the second, the presence of a hydrocephalus internus. In the third case, one of generalized convulsions predominating on the left side but with neurologic signs pointing to a right-sided lesion, the encephalogram suggested a focus on the left side as evidenced by distinct atrophy; here, therefore, encephalography added nothing of help in determining the etiology. The fourth case was one of jacksonian epilepsy in which, clinically, both the convulsions and the neurologic signs pointed toward a focus on the right side. Encephalography, on the other hand, suggested a lesion on the left side, and instead of helping, it tended to confuse the diagnosis. In the fifth and sixth cases the diagnosis of tumor, clinically suspected, was confirmed. (2) Intracranial tumors, six cases. In this group, encephalography helped materially in establishing the diagnosis of tumor, but was of no more value in localizing the lesion than a thorough clinical examination. (3) (*a*) Organic cerebral processes. Three cases in which the diagnosis of organic lesion was not altogether certain; encephalography helped in establishing the

organic basis of the process. (*b*) Dementia praecox. Encephalography showed marked atrophy in a case of long duration (the author considers the atrophy as effect rather than cause of the process). (*c*) One case of cerebral syphilis with encephalographic evidences of organic disease.

In summing up, the author comes to the conclusion that encephalography has given definite proof of its value as a diagnostic method. This value, however, should not be overestimated, and encephalography should remain an aid to rather than a substitute for clinical investigation.

MALAMUD, Foxborough, Mass.

METASTATIC CALCIFICATIONS IN THE ORGANS OF THE DOG AFTER INJECTIONS OF PARATHYROID EXTRACT. WILHELM HUEPER, Arch. Path. & Lab. Med. **3**:14 (Jan.) 1927.

Hueper investigated the calcium-magnesium relations in the body of dogs after injections of parathyroid extract. In the beginning, when the blood calcium had exceeded the normal level by from 2 to 3 mg., a psychic depression, a slight decrease of the pulse rate and an increase of the urine excretion were observed. With increasing blood calcium the symptoms grew more urgent. The dogs began to vomit in attacks, especially after intake of fluid, and the pulse rate dropped more markedly and became irregular. Simultaneously, the coagulation time showed a decrease from two minutes to one and one-half minutes. In the final stage, the animals vomited a bloody mucoid material or even pure blood more freely. The dogs died after a period of general weakness and dizziness with the symptoms of coma.

Autopsies indicate that parathyroid extract given in physiologic doses induces: (1) a prolongation of the systole and shortening of the diastole of the heart; (2) a dilatation of the vessels of the internal organs; (3) an intensification of the tonus of the stomach and intestine and (4) a rise in the calcium level of the blood.

The multiple hemorrhages in the brain are produced by stasis in the small veins and capillaries of the brain and represent secondary diapedetic extravasations. They were observed only in the brains of those dogs that died in coma, but were not seen in animals killed during an early stage of the hormone poisoning.

The authors conclude that: "Metastatic calcifications in various organs were produced by the injection of overdoses of parathyroid extract. The effect of the hormone on the action of the heart and the circulatory system is the cause of the hemorrhages, thromboses and secondary necroses in several organs. The extreme degree of calcium precipitation in the kidneys leads to a considerable impairment of the function of these organs, which may become fatal to the organism. The dangers connected with the therapeutic use of this hormone make a proper control of the blood calcium absolutely necessary."

COBB, Boston.

THE CONSTITUTION AND PHYSIQUE IN HEALTH AND DISEASE. F. VON ROHDEN, Arch. f. Psychiat. **79**:786 (Feb.) 1927.

The tendency to classify physique-character correlations into three groups has existed for a long time. Giovanni, Huter, Sigaud and Kretschmer were particularly prominent in these attempts. Huter recognized three types: (1) sensory (Empfindung), (2) motor and (3) nutritional, which correspond to Kretschmer's leptosome-asthenic, athletic and pyknotic types, respectively.

Huter advanced the theory that the disposition to types originates in the developmental preponderance of one of the three embryonic layers; preponderance of ectoderm predisposes to the development of a sensory type, that of mesoderm to motor, and that of endoderm to the nutritional type. The combined predominance of two of these layers will find expression in combined types; equality of the three makes for the harmonic (Kretschmer's noncharacteristic) person.

The author agrees with Huter that the disposition to develop into a certain type must be present in the embryo; the bodily structure is accompanied not only by a certain type of character but also by a predisposition to certain types of diseases, both physical and mental. He reports the results of studies in 1,319 cases. Of these, 382 were medically normal persons (criminals and students); 937 were mentally diseased persons (schizophrenic, manic-depressive, epileptic, feebleminded and general paralytic, and 128 examples of other types of psychoses). He concerns himself particularly with the three fundamental groups of leptosome, athletic and pyknotic; although he finds that in the normal persons these types show a relation of 64:30:6, he believes that his material is somewhat one sided and that normally they occur in the ratio: 60 leptosomes; 30 athletics; 10 pyknotics. In his pathologic material the most striking finding is that in the manic-depressive psychoses this relation is 10:3:87, showing an extreme preponderance of pyknotic persons. In epileptic persons, the relation is 33:67:0, i. e., an absence of pyknotic and a preponderance of athletic types. In the schizophrenic material, the author finds the relation 66:28:6; he brings this forward to substantiate Kretschmer's finding of a preponderance of leptosome-asthenic persons and a decrease in pyknotic persons. The author brings this as an argument against Kolle and Gruhle, who found that the relations of the three types in schizophrenia were practically the same as in normal persons. To the reviewer, the argument does not appear valid as the author's figures for normal persons are 64:30:6 and for schizophrenic persons, 66:28:6.

MALAMUD, Foxborough, Mass.

SYRINGOMYELIA WITH CAVITIES IN POSTERIOR NERVE ROOTS. J. D. COMRIE and J. W. DAWSON, Edinburgh M. J. **33**:566 (Sept. 19) 1926.

After summarizing the history of this disease from 1564 to the present time and describing Schlesinger's six types, the authors report a case of syringomyelia in which there were cavities in the posterior nerve roots. The patient was a woman, aged 51, who had had good health up to the age of 50; she had suffered with rheumatic fever at 14, with mitral stenosis as a residual symptom. The husband died from aortic disease, and his blood gave a four plus Wassermann reaction. Eight months prior to death, the patient complained of frequent micturition. About one month before death (one week before admission to the hospital), the patient complained of numbness in both legs and lassitude. Three days later, the left leg was weak and she was unable to walk. Urinary retention now developed. On admission to the hospital, one month prior to death, the following symptoms were noted: great loss of power in both lower emtremities; deep reflexes (knee and ankle) and plantar reflexes lost; Argyll Robertson pupils. Tactile sensation was present in the lower extremities but pain and temperature senses were lost below the middle of the thigh on both sides. The patient was unable to speak above a whisper. Four days prior to death, the upper extremities began to show involvement; trophic skin changes appeared, and death came suddenly. The Wassermann reaction with the serum was strongly positive. At necropsy, there was nothing of interest in

the body except patches in the aorta which were suggestive of syphilis. The brain was removed. Grossly, in the spinal cord there was a cavity extending from the second cervical segment to the first lumbar segment with its greatest cross section of area at the level of the seventh cervical segment. The posterior roots from the seventh to the tenth dorsal segment contained a separate cavity which grossly was not associated with the cavity in the cord. On histologic study, the fibers of the posterior columns showed the degeneration characteristic of tabes. The pia-arachnoid showed well marked, diffuse, and perivascular round and plasma cell infiltration. The cells of Clark's column in the dorsal cord could scarcely be recognized and at other levels were atrophic, pigmented and compressed. Helweg's tract in the first cervical segment was pale, and the fibers of Lissauer's zone were degenerated at all levels. The authors believe that the cavity in the cord in this case was secondary to syphilitic vascular disease. The article is accompanied by twenty photomicrographs and a short bibliography.

POTTER, Akron, O.

PRIMARY CARCINOMA OF THE BRAIN. H. KUFS, Arch. f. Psychiat. **78**:663 (Nov.) 1926.

On the basis of a clinico-anatomic study of a case of his own as well as on an analysis of cases previously reported, the author discusses the question of primary carcinomatosis of the brain. The case reported is one of multiple tumors of the brain. Three tumor masses are described: (1) in the left centrum semiovale; (2) in the posterior central convolutions; (3) in the right quadrigeminal body. None of these tumors had any definite relation to the ventricular system, but as the author had not, at that time, discovered any growth in the other organs that would suggest a primary tumor (some of the organs were examined histologically; in others, the macroscopically negative symptoms appeared to have satisfied the author), he concluded that he was dealing with a primary tumor of the brain. A further proof was offered by the structure of the growths, which even macroscopically presented the villous papillary appearance of previously described growths of this type, and which microscopically showed types of epithelial cells ranging from the high cylindrical ones in the younger villi and down to the flat cubic types in the older ones that are found in the ventricular and plexus epithelium. The malignant condition of the growth showed itself in its tendency to invasion and metastasis.

The report of a case of tumor of the brain of similar structure, but apparently a metastasis from a thyroid carcinoma, from the Aschoff Institute made the author change his opinion as to the nature of the tumors in his case. The negligence of the author to follow up the macroscopically negative thyroid by a microscopic examination precluded the possibility of definitely settling this particular point in his case. He comes to the conclusion, however, that the cases so far reported of so-called primary carcinomatosis of the brain originating in ependymal or plexus cells may be, after all, metastases of growths in some other organ not discovered at the examination.

MALAMUD, Foxborough, Mass.

CEREBRAL CONCUSSION. G. GAVIN MILLER, Arch. Surg. **14**:891 (April) 1927.

From the practical and the theoretical points of view Miller considers the differences between concussion and contusion of the brain and attempts to show that concussion may occur without contusion. He bases his definition of concussion on purely clinical and physiologic grounds: namely, that concussion should be used to indicate a group of symptoms which are the result of a

temporary inhibition of cortical function, with or without stimulation or inhibition of one or more of the medullary centers, but which are not accompanied by pathologic lesions. He attempts to correlate, by animal experimentation, physiologic phenomena of cerebral concussion with the accepted clinical symptoms of immediate unconsciousness with or without medullary effects which tend to progressive recovery or which may cause immediate death. The hypotheses of the etiology of concussion and its relationship to contusion are discussed at length. He seems to show by his experiments that a severe single blow or repeated light taps on the unbroken skull and electrical stimulation of the medulla or direct mechanical pressure on the medulla all produce one and the same physiologic effect on respiration and blood pressure. He does not believe that cerebral anemia is a factor in the production of the clinical or physiologic symptoms of concussion.

He does not believe that contusion always accompanies concussion because contusion implies death of the cells, whereas since concussion may be followed by recovery, the functions of the medullary centers have merely been momentarily paralyzed and not permanently damaged. He feels that he has shown both by argument and experiment that the term concussion should be used to denote the generalized paralytic effect of an impact on the cortex and medulla and should be distinguished from the term contusion, whether a gross or microscopic contusion is meant, and also from the term hemorrhage, whether reference is made to a diffuse or petechial hemorrhage.

GRANT, Philadelphia.

HEREDITARY INFLUENCES IN POSTENCEPHALITIC PSYCHIC DISTURBANCES. U. FLECK, Arch. f. Psychiat. **79**:509 (Feb.) 1927.

The results of a study of hereditary data in fifty-two cases of metencephalitis showing psychic disturbances are presented. The main thesis followed by the author deals with the possibility of discovering whether the frequent occurrence of psychopathic trends in the family tree tends to determine the nature of the postencephalitic sequelae. In the fifty-two cases, thirty-five patients were below the age of 20. All the cases showed some mental deviations, twenty-seven of them representing the different types of psychopathies. Practically all of the psychopathies were found among the patients under 20 years of age. The study of the family trees of these persons included siblings of the patient and their parents and grandparents. Mental deviations noted were those of asocial tendencies, psychopathies, schizophrenias and manic-depressive psychoses.

The results can be summarized as follows: 1. Most postencephalitic patients below the age of 20 with psychopathic character changes showed a much greater frequency of psychopathic traits in the family tree than the older postencephalitic or normal individuals (the latter two being about equal). 2. This and other studies showed that psychopathic traits in the families of normal persons, as well as those who were subject to influenza but who had not developed encephalitis and also those who had developed encephalitis but had not shown any psychopathic manifestations following it, are about equally represented. Thus, heredity does not play any definite rôle in predisposing a person to encephalitis or its sequelae other than psychopathies. 3. An analysis of the type and intensity of the psychopathic traits presented in this work would point to the fact that the frequent occurrence of psychopathic traits in the family is of a more pathoplastic than pathogenetic importance. It is the nature of the psychopathy rather than its occurrence that seems to be predetermined by hereditary influences.

MALAMUD, Foxborough, Mass.

SOME CHANGES IN THE CEREBELLUM IN A CASE OF DEMENTIA PRAECOX. S. RAMÓN Y CAJAL, Trav. d. lab. d. rech. d. l'Univ. d. Madrid **24**:181 (Dec.) 1926.

The case reported is that of a man, aged 65, with dementia praecox, who died of uremia. Cajal is careful to point out that the changes in the cerebellum which he describes cannot be ascribed to the dementia praecox, but are more probably related to a senile dementia. They are to be regarded, in part, as degenerative, and in part as regenerative, changes in the neurons. They include: (1) the development of a thin process arising from the soma of a Purkinje cell, which resembles the primordial processes, is pale and appears atrophic. It thins out in the middle of its course, being sometimes very long and tangentially directed, then thickens suddenly and spreads out into a star-shaped body without a nucleus, but possessing an indistinct neurofibrillar net. (2) Localized, fusiform, triangular or star-shaped hypertrophies of the dendrons of Purkinje cells. They occur usually in the course of the main dendron and may be so large as to resemble a displaced cell body. Smaller masses also occur at the point of bifurcation of dendrons of the second or third order. (3) Recurrent terminal dendritic bulbs. (4) Aberrant dendritic arborizations showing curious anomalies in course, such as curving back on themselves in an arc and peculiar sinuous formations that resemble snake; they end in various layers of the cortex with terminal knobs or fine branches. (5) Disappearance of Purkinje cells. (6) Changes in the pericellular nests. Similar changes have been described in various other conditions, notably in general paralysis and senile dementia.

SINGER, Chicago.

STUDIES ON THE ANTERIOR LOBE OF THE PITUITARY BODY. W. S. JOHNS, T. O. O'MULVENEY, E. B. POTTS and N. B. LAUGHTON, Am. J. Physiol. **80**:100 (March) 1927.

The authors report carefully controlled experiments relative to the effect in dogs of intramuscular and intravenous injections of protein-free extracts prepared from the anterior lobes of fresh ox pituitary. They find that within twenty-four hours striking hyperglycemia, polyuria and glycosuria become manifest with a temporary decrease in the first two reactions after a few days, followed by a second rise which continues as long as the extract is administered and after its discontinuance subsides in several days to the original levels. It is felt that the hyperglycemia depends on increased glycogenolytic activity, since the livers of treated dogs show practically no glycogen and also, as shown in previously treated animals, on the administration of a second course, glycosuria and polyuria follow without evidence of hyperglycemia. The authors suggest further that anterior pituitary exerts a lowering effect on the renal sugar threshold. In support of this is the persistence of glycosuria in the presence of a diminished blood sugar content and volume of urine during the administration of the extract, and the occurrence in previously treated animals of glycosuria and polyuria in the absence of hyperglycemia. Unlike "pituitrin" the anterior lobe extract produces only a slight rise in blood pressure and exerts no contraction effect on strips of uterus from a fresh rabbit or guinea-pig.

RAPHAEL, Ann Arbor, Mich.

SPINAL CORD IN TETANUS. S. H. NELSON, Lancet **2**:852 (Oct. 23) 1926.

The author reports briefly a case of tetanus in a girl, aged 16, who developed symptoms sixteen days after a puncture wound on the left foot. She was admitted to the hospital fourteen days after the puncture wound. Death

occurred twenty-one days after the injury in spite of subcutaneous, intravenous and intrathecal treatment with tetanus antitoxin. At necropsy, the organs of the body showed nothing of interest and the cord only was preserved. After fixation in alcohol, sections from the cord and root ganglia were studied. The sections from various levels of the cord showed a congestion of the vessels and small hemorrhages in the posterior columns. The anterior horn cells showed various degrees of change with some eccentric nuclei. The cells of Clarke's column showed marked chromatolysis and eccentricity of the nuclei. In the sections from the ganglia of the posterior roots there was a lack of the internal differentiation of the two types of cells. The nucleolus was distinguished with difficulty in all the nerve cells. There was some pyknosis. Neuronophagy was present but not marked. The author believes that these observations warrant the deduction that the histologic changes in the cord and in the ganglia of the posterior roots may be attributed directly to the action of the tetanus toxin.

POTTER, Akron, O.

STUDIES OF BASAL METABOLISM IN NEW ORLEANS. H. HAFKESBRING and P. BORGSTROM, Am. J. Physiol. **79**:221 (Dec.) 1926.

Careful studies of basal metabolism were carried out by the authors on a series of nine normal men and women. It appears from their results that the metabolic rate in these subjects from New Orleans is definitely lower than the range determined by northern workers and generally accepted as standard. The New Orleans figures averaged 16 per cent below the average standards established by DuBois, Harris-Benedict and Dreyer. This work is of real significance, bearing importantly on the general question of metabolism in tropical and subtropical climates, which it seems, from other reports also, exert a definitely lowering influence. This, if true, represents a critical point with reference to the clinical interpretation of results determined in such climates. The authors also find that the metabolic rate shows a tendency to vary in relation to the length of the fast period following the last meal, reaching a minimum in from the fifteenth to the nineteenth hour, after which a sharp rise occurs. Likewise there is some fluctuation dependent on the quantity and quality of the food taken at the last meal. These facts likewise are of practical clinical importance and suggest the advisability of appropriate modification in the routine of the standard test.

RAPHAEL, Ann Arbor, Mich.

THE HERTWIG-MAGENDIE PHENOMENON. H. OLOFF and H. KORBSCH, Arch. f. Psychiat. **79**:200 (Dec.) 1926.

The authors describe a case of tumor of the brain which, among other symptoms, showed the Hertwig-Magendie deviation; they discuss the mechanism and possible localization of the phenomenon. The phenomenon consists of a disturbance of simultaneous movement of both eyes. The eye of the diseased side points downward, the other upward, and frequently there is also a lateral deviation, both eyes looking away from the lesion.

The case reported was that of a tumor of the brain in which the deviation was manifest two months before death. Autopsy revealed an infiltrating glioma of the right cerebellar hemisphere and its peduncles. Following the presentation of an exhaustive histopathologic study of the material, the authors come to the conclusion that, with Poetzetl and Sittig, they would localize the lesion responsible for the phenomenon at about the corpora quadrigemina. They believe that in such cases one is dealing with a disturbance of a mechanism associated with the tracts running from the vestibular nuclei over the posterior

longitudinal bundle to the oculomotor nuclei. An analysis of this case, as well as the others reported in the literature, pointed to the fact that the posterior longitudinal bundle is the one most usually affected in such cases.

MALAMUD, Foxborough, Mass.

PHOTOGRAPHIC DEMONSTRATION OF CERTAIN PHENOMENA OF NERVE REGENERATION. S. RAMÓN Y CAJAL, Trav. d. lab. d. rech. de l'Univ. d. Madrid **24**:191 (Dec.) 1926.

After commenting on the technical difficulties of securing photographs from the thick sections necessary for the study of the regeneration of the nerve, Cajal presents excellent photomicrographs from thinner sections (from 15 to 20 microns), selected from a large number collected during many years, which demonstrate with clearness that: (1) the neural outgrowths in the scar and in the peripheral end of the divided nerve arise from the axons of the central end; (2) every early exploratory fiber travels freely in its path of growth—the individual or collective sheath is developed later, six or seven days after the operation; (3) the free extremity of the outgrowths in the ameboid phase may assume various forms, including that of terminal sheaths spread over obstacles, both when they travel inside tubes of Schwann and when they are lost in the mesodermal tissues; (4) under favorable conditions, innervation of the peripheral end, as well as of ingrafted segments of nerve, can be observed prior to the differentiation of the bands of Büngner (five days after operation).

SINGER, Chicago.

SOME RESULTS OF A SECOND INDUCTION OF MALARIA IN GENERAL PARALYSIS OF THE INSANE. J. ERNEST NICOLE and JOHN P. STEEL, J. Neurol. & Psychopath. **7**:208 (Jan.) 1927.

Twenty patients were given malaria a second time, on an average of seventeen months after the first induced attack. Mosquito infection was found more effective in producing the second attacks than the injection of a patient's blood. Eight months' observation show: one patient dead; seven unimproved; seven slightly improved; two have made marked progress; three have been discharged. In view of the fact that these cases showed little or no improvement after one attack and twelve of the twenty showed improvement on reinoculation, it seems worth while to renew malarial treatment of general paralysis in suitable cases.

FAVILL, Chicago.

PATHOLOGY OF THE CENTRAL NERVOUS SYSTEM IN MALIGNANT TERTIAN MALARIA. J. G. THOMPSON and S. ANNECKE, J. Trop. Med. **29**:343 (Oct. 15) 1926.

Thompson and Annecke report the study of the central nervous system in twelve cases of malignant tertian malaria, in which death was directly due to coma. Sections of the nerve tissue had a smoky, gray appearance, with edema, hyperemia and punctiform hemorrhages. In the microscopic sections in three cases, nodules (malarial granulomas) were found in the cerebrum and one in the cerebellum. These so-called nodules contained a central capillary filled with parrasitized red cells, surrounded by a perivascular necrotic area with proliferating glia cells and red blood cells without parasites. The subarachnoid membrane showed diffuse perivascular small celled infiltration. The chief histologic manifestation was a more or less complete blocking of the capillaries with parasitized red blood cells, resulting in edema, punctiform hemorrhages and thrombosis.

POTTER, Akron, O.

MENINGOBLASTOMA OF THE CHOROID PLEXUS. CHARLES OBERLING, Ann. d'anat. path. et d'anat. norm. **4**:379 (April) 1927.

The tumor was found fortuitously at autopsy in a woman, aged 72, who died of chronic nephritis. It was indefinitely spherical, about 4 cm. in diameter, and occupied the posterior horn of the left lateral ventricle. The walls were considerably depressed, but the brain was not seriously deformed. Instead of finding the epithelial neoplasm as was expected, the picture bore many resemblances to the older meningeal type of tumor, even to containing concretions. The formation of whorls and the presence of obliterated vessels rendered this resemblance even more striking. The origin is explained on the supposition that certain meningoblasts were included in the stroma of the choroid plexus.

FREEMAN, Washington, D. C.

Society Transactions

NEW YORK NEUROLOGICAL SOCIETY

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LOUIS A. CASAMAJOR, M.D., *Vice-President, in the Chair*

DEEP PAIN SENSE IN TABES DORSALIS. DR. JUNIUS STEPHENSON.

By the deep pain sense is meant the deep pressure pain sense of the calf muscles. The diminution of this sensation in one of the calf muscles is considered the first practical objective observation in the majority of cases of tabes dorsalis. It is agreed that alteration in the vibratory fields is the first morbid change, but this is not considered practical from the standpoint of the general practitioner to whom the early tabetic patient first goes for relief, as few of them have tuning forks, and if so, the instrument would not be sufficiently acute to demonstrate the early changes. It is contended that the sequence of behavior in the early development of tabes dorsalis is as follows: First there would be an alteration of this sensation in one group of calf muscles, then changes in the ankle jerk of the same side after which the same sequence would follow in the other leg. The author felt that by the time the knee jerks are not elicitable definite spinal cord pathologic change is present. It was stated that in disease conditions known to have their early morbid changes in the cord, such as subacute combined sclerosis, this phenomenon is rarely or ever present; this argues for the peripheral origin of tabes dorsalis. The theories of the meningitic and neuritic or radicular origin were held tenable, and it was advanced that the behavior of the deep pain sense in comparison with the condition of the ankle jerk supported one or the other of these processes.

In certain cases there is an early and profound alteration in the deep pain sense with slight, if any, change in the ankle jerk of that side. In other cases, whereas the involvement of the deep pain sense is present, it is out of proportion to what might be expected in comparison with the considerable alteration of the ankle jerk. The explanation of this behavior was as follows: In the former the original process is a meningitis with pial constriction of the roots producing two opposed effects, one an irritant, causing the shooting pains, the other a decrement, causing a block in the conveyance of the deep pain sense. For this reason the opinion was advanced that ultimately the bundles conveying this particular sensation are located near the periphery, and hence are the first to feel the constriction produced by the inflammatory changes in the pia. There may be a vague action of the toxin of syphilis on the root in this type, but the other chief factor is the element of compression. In the second case the great incidence of the toxin is within the root itself, and being focussed therein involved more of the structures, hence more involvement of the ankle jerk.

No pathologic proof of this interpretation was presented, but suggestions were invited. The phenomenon seems to be an entity, and it was hoped that a discussion of this early sign might prevent many tabetic tragedies.

DISCUSSION

DR. WALTER KRAUS: I think that the reason Dr. Stephenson asked me to discuss this paper was that he recalled that during the war and shortly thereafter I was much interested in the functions of the fasciculi which constitute peripheral nerves and roots. Under the guidance of Dr. Charles H. Frazier and in collaboration with Dr. Samuel Ingham, a good deal of work was carried out in this direction on the peripheral nerves and the brachial plexus. We did not get much nearer a

solution of the problem than did Sir Charles Bell in 1811. It is interesting that Ingham and I, publishing our results more than one hundred years after Bell, felt that we did not know much more about the functions of the fasciculi than he did.

Any thesis regarding the effect of constriction of the roots in contrast to the effect of a parenchymatous lesion is extremely difficult to maintain, for the simple reason that we have no knowledge of the anatomic functions of fibers within the roots. However, I think that Dr. Stephenson's idea may be used as an explanation for the presence of a sensation of pain in the absence of reflexes. In disease of the thalamus, pons, spinal cord roots, and peripheral nerves there may be an immense amount of hyperesthesia and subjective pain. The lightning pains of tabes belong in this group.

All of the severe pains in disease of the pathways of pain leading into the thalmus are properly called causalgias. Not only will irritation of the nerve fibers inside or outside the central nervous system produce this causalgia, but interruption may also bring about the same result. The central stumps and fibers will be irritated by a scar. In a constricting lesion, the knee jerk will be absent because it is a fragile reflex; at the same time, due to irritation, the patient will be extremely sensitive to squeezing of the achilles tendon. In a parenchymatous lesion both reflexes and the sensation of pain may be absent. I think that it is important for us to examine patients with tabes dorsalis for this subtle difference which Dr. Stephenson has made, and then if we are so fortunate as to obtain the spinal cord post mortem, we may make certain whether Dr. Stephenson's thesis that a parenchymatous lesion of the root will produce one syndrome and a constricting meningitis will produce another is borne out pathologically or not.

THE CONNECTIONS OF THE PARS SUBOCULOMOTORIA OF THE SUBSTANTIA NIGRA. ARMANDO FERRARO, M.D.

The substantia nigra has acquired great importance since the development of the studies on the extrapyramidal system. Although Vogt was still uncertain as to whether the substantia nigra belonged to the extrapyramidal system, further studies have undoubtedly established this relationship. Monakow, who took especially into consideration the cortical connections of the substantia nigra, believes that this formation depends on the sigmoid gyrus and third frontal convolution. The dependence of the substantia nigra on the cortex was supposed by Monakow to be of the same type as is the case with the optic thalamus, meaning that the axons of its cells reach the cerebral cortex. Monakow's views were almost generally admitted, and even Äriens Kappers, in the first edition of his fundamental work on comparative anatomy, subscribed to the same idea. Here and there, however, an anatomic suggestion is found that the substantia nigra may be connected with the basal ganglia (Tarassewitch, G. Holmes, Jakob, etc.). Among others, Langley and Grünbaum explicitly state that following the removal of the basal ganglia only a few nerve cells of the collection remained in the mesial region of the substantia nigra, while this region was normal in a case in which only the cortex was removed.

Mirto believes that the axons of the substantia nigra are sent into the tegmentum, although he was not able to specify where they end. Cajal thinks that they cross the midline to reach the opposite side, while Amaldi believes that these axons are directed toward both the tegmentum and the pes pedunculi. Mingazzini also believes that they go downward to the pes pedunculi, a direction which is also admitted by Karplus and Spitzer through their "Grenzbundel der Pyramide." Bauer states that the axons of the nigra go to the tegmentum where they connect this formation with other areas of the central nervous system by means of fibers which he calls "fibrae efferentes substantia nigrae." Similar fibers have also been described by Marburg.

Connections established through fibers originating in the cortex and ending in the substantia nigra were admitted a long time ago by Meynert, Mingazzini,

Dejerine, Obersteiner, and others, and recently Minkowski, Jakob, Foix and Nicolesco have emphasized their importance. Connections have also been established between the corpus striatum and the substantia nigra by way of nerve fibers going from the striatum to the substantia nigra. One important pathway for this connection is the "Kammsystem des Fusses" or tractus striopeduncularis of Edinger. Wilson mentions also nerve fibers going from the pallidum to the substantia nigra and the same connections are admitted by Vogt, Wallenberg and Jakob. Connections between the corpus of Luys and the substantia nigra have also been found by Bauer, Marburg and by myself (fibrae subthalamicae—substantia nigra). Besta and Jakob speak of connections between the thalamus and the substantia nigra, while Hatschek and Schlesinger mention the connections of the nigra with the lateral pontile nuclei.

Jurmann and Bechterew admit that there are connections between the substantia nigra and the posterior white columns of the cord by means of fibers from the nuclei gracilis and cuneatus. Kölliker speaks of fiber connections between the internal geniculate ganglion and the substantia nigra, and Wechsler's observations confirm those of Kölliker.

Connections between the corpus geniculatum laterale and the substantia nigra have been reported by Ziehen by the mean of its "Kometenbundel," and Ziehen, Ganser, Wallenberg and I have found connections between the pedunculus corporis mamillaris and the substantia nigra. Sterzi believed in direct connections between the substantia nigra and the tractus peduncularis transversus, while Costaldi thinks that the connections are indirectly established by means of the axons of the nucleus tractus peduncularis transversus.

In order to elucidate the question of the connections of the substantia nigra with the higher structure on which it is dependent, I have performed several different series of experiments in cats, dogs and rabbits. In the first series I have removed the frontal region and the gyrus sigmoideus of the cortex. In a second series I have removed the whole cortex, and in a third series I have removed the cortex plus the corpus striatum. The results of my experiments may be summarized as follows: (1) After removal of the frontal and sigmoid region, no disappearance of the substantia nigra was found. This formation was smaller than the normal, but no noticeable disappearance of nerve cells could be detected. (2) Following removal of the whole cortex, no disappearance of the substantia nigra was noticed. The whole structure was markedly reduced in size, but this fact may be attributed to the degeneration of the pes pedunculi and to the disappearance of a certain amount of the stratum molecularis intercellularis. The nerve cells of the substantia nigra were slightly diminished in size, but noticeable disappearance of them could not be detected. (3) Following the removal of the cortex and of the neostriatum (nucleus caudatus and putamen) there was a definite disappearance of cells of the substantia nigra; not localized but diffusely distributed in the entire structure. The disappearance of the substantia nigra was proportionate to the removal of the neostriatum. The removal of the neostriatum and paleostriatum was followed by an almost total disappearance of the substantia nigra. There were, however, a few nerve cells which were preserved in this formation even after removal of the whole cortex and striatum.

I concluded, from these experiments, that the majority of the axons of the substantia nigra do not go to the cortex, as Monakow and others believe, but that they do go to the corpus striatum, the pallidal connection seeming to be of greater importance. It is therefore necessary to refute the conception of the substantia nigra as being dependent on the cortex ("Grosshirnanteil") and substitute for it the conception of dependency of the substantia nigra on the corpus striatum.

I wish to emphasize that I do not deny the connections of the cortex with the substantia nigra. I do admit them and believe them to be important, but these connections are not connections of dependency; in other words, the corticonigric fibers constitute, so to speak, an afferent pathway of a reflex which presumably

takes place in the substantia nigra, the efferent pathway of which is represented by the nigrostriate nerve fibers.

In my previous studies of the substantia nigra, of which I have summarized the results, I was struck by the presence, in the mesencephalon of the cat, of a special collection of nerve cells which I found located between the nucleus of the third nerve and the mesial portion of the substantia nigra. This group of cells is a bilateral one, situated on each side of the midline, and has an oblique course directed dorsoventrally and from within outward. This group was first mentioned by me as a nucleus linearis suboculomotorius.

In order to establish the connections of this special group of cells, I have performed a new series of experiments on cats, following the same schema as that traced in the previous part of this note. The conclusions I reached may be summarized as follows: 1. Removal of the whole cortex does not affect this special formation. 2. Removal of the cortex plus a portion of the corpus striatum partially affects this group of cells as a certain number of them disappear. 3. Total removal of the cortex and of the striatum is followed by an almost entire disappearance of this group of cells.

I feel justified, then, in concluding that the connections of this special group of cells are fundamentally the same as those of the substantia nigra, and as this group is in continuity with the main part of the substantia nigra, I drew the conclusion that this group of cells must be considered as a mesial prolongation of the substantia nigra which reaches the ventral portion of the nucleus oculomotorius.

I tried to locate the same nucleus in man, but, lacking serial section material, I was able to establish between the substantia nigra and the nucleus oculomotorius only the presence of pigmented nerve cells which are disposed in a lineal and oblique direction as the pars suboculomotoria which I have described in the cat. In order to be sure of the relationship of these cells to the substantia nigra, I tried to trace these cells in cases of chronic epidemic encephalitis. I was able to establish that in connection with the disappearance of the main portion of substantia nigra the cells of this lineal portion were also absent.

Altogether I feel justified in believing that the lineal group of cells which I found in the cat belongs to the substantia nigra of which they represent a simple mesial prolongation. As far as the function of this special portion of the substantia nigra is concerned, I can say hardly anything, as the functions of the main portion of the substantia nigra are still debatable. I may, however, advance the hypothesis that, owing to its close relationship to the nucleus oculomotorius, a correlation might be established between the muscular tonus of which the substantia nigra is supposed to be a regulating center and the ocular movements.

DISCUSSION

Dr Walter Kraus: Is the neucleus cupuliformis, which lies at the caudal end of the red nucleus, related to the nucleus suboculomotorius?

Dr. Ferraro: There is no connection between the two.

Dr. Louis Casamajor: This paper is of interest from many points of view. One is the question of the relation of this particular study to the pathology of epidemic encephalitis, as to whether the disappearance of the cells in the substantia nigra might be secondary to a disturbance in the striate body. I wonder just what this disappearance of cells means. I know of no other place in the nervous system in which simple injury to the fiber causes disappearance of the cell, and I wonder whether that is not more a matter of straining than a real loss of cells. In amputation of a limb studies show that the cells do not tend to disappear from the ventral horn. They remain throughout life in the condition of central chromatolysis. Do these cells disappear because the end of the axons is cut so that they can no longer function as nerve cells? Possibly the nerve impulse flow is the

other way, and these cells are altered by the fact that they are cut off from their natural stimulus from above. Dr. Ferraro gave the impression that the flow was from the substantia nigra to the latter developed structures of the striatum.

DR. FERRARO: From the clinical point of view in encephalitis, I have asked myself if some of the cells which disappear in the chronic stage of encephalitis disappear as the result of a lesion of the striate body. I do believe that a few of them may disappear following the specific lesions of the striate body, but as a fact, in epidemic encephalitis, I am more inclined to believe that since the substantia nigra is a seat of election for the infection, its destruction is more primary than secondary. It would be more likely to be a secondary destruction if such cases as Wilson's disease, pseudobulbar paralysis or any other process in which there are areas of softening of the striate body are considered. In these cases the disappearance of cells may be correlated with the destruction of the corpus striatum. The reason that the cells of the substantia nigra disappear when the striate is removed is that the principles of the law of Gudden are followed. Retrograde degeneration of the cells follows a different rule in the new-born, the young and in adult animals. I have operated on young animals. In those cases the law of Gudden holds true; following section of the axon, there is a true disappearance of the cell from which the axon originated. It is in accordance with this principle that Monakow, Nissl and Gudden himself have based their fundamental studies on the structure of the nervous system (connections of the thalamus, red nucleus, and corpus geniculatum laterale.) I believe that in my cases there was a real disappearance of cells and not a loss of stainable properties.

CEREBRAL CICATRIX: THE REACTION OF NEUROGLIA TO BRAIN WOUNDS. DR. P. DEL RIO-HORTEGA and DR. WILDER PENFIELD. (From the Laboratorio de Histopathologia de la Junta para applicacion de Estudios, Instituto Cajal, Madrid.)

The study is an experimental one devoted to the change of neuroglia astrocytes about brain wounds. The authors pointed out, however, that microglia cells play an important active part in the wound itself. In the early days after the infliction of the wound, all transitions could be seen from ramified spider-like microglia cells, such as are seen in the normal cerebrum, to compound granular corpuscles in a zone immediately about the wound. Mitotic division of these cellular corpuscles at the wound margin began as early as the third day. After the products of destruction had been cleared away, as for example on the fifty-second day after a small stab wound in a rabbit's brain, microglia cells appeared in the scar itself in a complicated form resembling those seen normally in the cortex of the brain. No evidence was seen of transition from neuroglia astrocytes or oligodendroglia to compound granular corpuscles.

It was pointed out that oligodendroglia plays no part, so far as could be seen, in the wound reaction. As a result of direct injury these cells seem to disappear as do the nerve cells.

Neuroglia astrocytes pass through definite phases:

1. *Swelling.*—At the end of twenty-four hours the astrocytes in the margin of the wound are swollen. On the third day it can be seen that the astrocytes closest to the wound are irregularly swollen and the expansions are fragmented. They are undergoing the destructive ameboid change of Alzheimer or clasmatodendrosis and contain coarse granules. Farther removed from the wound the cells are diffusely swollen and contain fine granules. The latter change precedes multiplication.

2. *Division.*—As early as the third and fourth day after a simple wound, the astrocytes, the nuclei of which have enlarged, show evidence of direct division. Each of the daughter cells takes to itself certain of the parent expansions. No evidence of mitotic division of neuroglia is seen.

3. *Fibrillation.*—Regardless of whether the astrocyte was protoplasmic or fibrous before the wound was made, the astrocytes on the border of the wound lay down fibers. By the nineteenth day a well defined zone of hypertrophied fibrous astrocytes is seen and a fairly sharp line of demarcation from the astrocytes of the brain. With the formation of fibers the astrocytes take on an arrangement like the spokes of a wheel. The larger expansions radiated toward the center of the wound.

4. *Contraction.*—Small stab wounds almost invariably contain connective tissue at their core and in it a plexus of newly formed vessels. New collagen fibers are numerous. It is about such wounds that the radiating arrangement of astrocytes is seen. Occasionally a stab wound remains gaping in part. This is particularly true in the white matter. In such a case there is no tendency to a radial arrangement.

It is pointed out that in spite of the careful study of the stages of astrocyte change, using selective methods which demonstrate these cells with great clearness, there was no evidence of any transformation of these cells into mobile macrophages. It is felt that such a transition can be practically ruled out in brain wounds. Many macrophages were present, but their presence could be adequately explained by derivation from microglia and possibly from the adventitial cells of the vessels. Increase in number of such cells is provided by their own mitotic multiplication at the site of maximum cerebral destruction. Thus the activity of these compound granular corpuscles in the wound resembles that of macrophages about areas of destruction elsewhere in the body, and they evidently have the same mesodermal origin in the central nervous system as in other organs of the body.

DISCUSSION

DR. CHARLES A. ELSBERG: There is little to say concerning the interesting histologic study that Dr. Penfield has made. It opens up a new pathologic method of research into the convulsive states and the condition which we call epilepsy. It would be interesting to hear from Dr. Penfield whether he has had occasion to study the brains of epileptic persons in order to determine whether changes in the astrocytes, along the lines he has described in these brain wounds, have occurred as an indication of continual irritation of certain portions of the brain through the tensile pull of the changing astrocytes, both in the traumatic and in the so-called idiopathic epilepsy, and whether the old ideas of gliosis of Alzheimer and others have any connection with the changes he has described in these brain wounds.

DR. J. H. GLOBUS: This is but one more demonstration of the excellent work of Dr. Penfield. Dr. Penfield has been exceedingly generous in telling us all that he has learned in Spain and in teaching us abundantly and clearly. It is interesting to note that a process similar to that in experimentally produced brain scars, is seen in the healing of spontaneously produced brain wounds. Practically every stage he described in the experimental material can be seen in spontaneous lesions of this kind, which may be due to vascular or inflammatory disease. There is only one question that I wish to ask Dr. Penfield: Can he be absolutely certain that oligodendroglia does not participate in the transition of the glial elements into granular cells? Can he be sure that this type of glia cell, giving off a short process here and there, does not form gitter cells? I think I have had sufficient experience, however, to agree with him that the other forms of glia, the astrocytes, do not take part in the formation of gitter cells.

DR. LEWIS STEVENSON: I would like to hear a little more about the microglia. It seems to me that the most dramatic thing in the healing of a brain injury is the tremendous migration of microglia cells to the aid of the brain which has been wounded. This has been worked out by Dr. Hortega, and his preparations of the microglia, starting from the normal cell and showing all stages of transition, just

as Dr. Penfield showed you in his drawing, are fascinating. I think they leave no doubt that these cells are microglia, that they are a third element in the nervous system and that they are of mesodermal origin. These microglia cells remove all the debris and take it to the subarachnoid space. Dr. Penfield has taken up the description of the process after this most dramatic event has occurred.

The staining method of Rio Hortega, the silver carbonate method, has made these studies possible. I do not think they were possible before, and I believe it is opening a great field not only in neuropathology, but also in general pathology.

Dr. Ferraro: Dr. Penfield and Dr. Globus have agreed that the microglia is the only responsible factor for the formation of the granular cells, and that astrocytes do not participate in this formation. I do not think that such a statement can be fully sustained. I admit, too, that the microglia has a large importance in the formation of the granular cells, but it cannot be denied that the astrocytes also contribute to their formation. There are instances in which one can easily follow the transition from the normal astrocyte through several stages to the formation of gitter cells, and I think that it is in this way that Dr. Hortega found the transition of his microglia into granular cells. If it is accepted that morphologic transformation occurs it cannot be denied that the astrocytes do participate in the formation of gitter cells. I have cases of encephalitis periaxialis diffusa in which the fact is clearly demonstrable. Of course, there is a microglial reaction, but there is also an intense reaction of the common type of the neuroglia, with a gradual transition into fatty granular cells. If the astrocyte had nothing to do with the granular cells, I would like to ask how Dr. Penfield can explain the transformation of the fixed type of Abbau into a mobile one with final formation of gitter cells. I do not feel that it is possible to exclude the participation of the astrocyte in the formation of the reticular cells.

Dr. Casamajor: Dr. Penfield has well proved the point that he started to make. This description of the healing of wounds of the brain follows the lines described in the past; Dr. Penfield has shown in this group of slides the series of changes in the process. The origin of microglia cells is purely of academic interest. It interested me that Dr. Ferraro should make the differentiation he does. I have felt certain that the oligodendroglia participates, and now that Dr. Ferraro has said that the astrocytes also are involved, I think we are coming back to the view that Alzheimer held, although the various classifications that Rio Hortega has offered are surely a permanent part of the nomenclature. Those pictures of the astrocytes certainly are important in demonstrating the connection between astrocytes and mesodermal tissues, especially the blood vessels, so that the concept of the astrocyte standing as an intermediary between the blood vascular system and the nerve tissues is well borne out when one sees the tenacity with which the astrocytes adhere to the blood vessels and other tissues of mesodermal origin. The question of pull and strain, which Dr. Penfield mentions, must certainly play a part in our pathologic concepts of the nerve tissues. Dr. Elsberg's remark that here are the possibilities of explaining some of the rather remarkable consequences of wounds and injuries of the brain that appear on the surface to have no connection anatomically with the part affected, is material for thought. I wonder if we are not going to open up some new ideas in the pathology of the brain, and perhaps get a little light on the pathogenesis of common nervous diseases concerning which we are at present so much in the dark.

Dr. Penfield: As Dr. Casamajor has pointed out, much of this work has been done before. Most of the stages of astrocyte change, particularly, have been described. We have endeavored to present the complete picture.

The application of the work to epilepsy of which Dr. Elsberg speaks is of importance, but I know nothing about it from a practical point of view. The work of Sze, of Dandy and of Elsberg shows that if a simple experimental wound is made in the brain of an animal, the dose of convulsant necessary to produce an

epileptic seizure is greatly reduced; it has occurred to me that possibly the factor of local contraction and pull on the vessels at a distance may help to explain the alteration in the threshold for convulsion.

Dr. Globus spoke of the possibility of transition of oligodendroglia into compound granular corpuscles. One has no right to say that one type of cell never develops into another. Nothing is more difficult than to be sure of transitional stages, and surely many errors have been made in pathology and histology on the basis of fancied transitions. I have never seen what I thought was transition of oligodendroglia into compound granular corpuscles, and the most striking evidence against such a metamorphosis is the reaction of acute swelling of oligodendroglia either post mortem or ante mortem. This process causes oligodendroglia to swell throughout the brain, but there is no production of compound granular corpuscles. Of that I am sure, as Dr. Cone and I have produced the phenomenon at will and have seen clearly every phase. Postmortem autolysis does not produce change in the microglia up to forty-eight hours or even three days after death, whereas the oligodendroglia cells are all changed. About a wound of the brain the oligodendroglia cells close to the margin are normal in appearance, while the microglia shows a local alteration. If the animal is allowed to lie for a certain length of time after death, and if opportunity is given for postmortem autolysis, the oligodendroglia becomes swollen throughout the brain, but not more so near the wound.

Dr. Ferraro spoke of the formation of compound granular corpuscles; this question is a battleground just now in neurologic pathology. He has voiced the attitude of the German school as I understand it; in their recent work on neuroglia they seem to adhere to contradictory points of view. For instance, Metz and Spatz agree that compound granular corpuscles are formed from microglia, and they think these compound corpuscles are mobile. They showed that iron pigment in general paralysis is found only in the microglia. They go on to say further, perhaps in an effort to appease the shade of Alzheimer, that astrocytes also form compound granular corpuscles, but that the latter corpuscles are not mobile. Thus they must believe in two types of cells, a mobile and a fixed compound granular corpuscle.

Evidently there is a confusion in the definition of these cells. Certainly neuroglia astrocytes are found with phagocyted substance in their cytoplasm, particularly when these cells are in a state of chronic degeneration. Some of the contained substances may have come from without, while other granules are the product of autodegeneration. Other types of fixed cells also are phagocytic, including nerve cells, as has been shown by the ingestion of lamp black. Neuroglia cells likewise ingest lamp black, but the fact that these astrocytes are phagocytic in one sense, does not mean that they are the mobile compound granular corpuscles with which we are so familiar. Metchnikoff pointed out the difference long ago between macrophage and ingestion of substance by fixed cells.

In certain nonspecific stains it is impossible to say that there is no transition between the swollen astrocyte and the microglia cell. In reading Alzheimer carefully it will be found that he sometimes says that the cells going to work on the products of destruction are fixed; at other times, he seems to say astrocytes are transformed into wandering macrophages. If the mesodermal origin of microglia is accepted, then are phagocytes in the brain comparable to the cells which become active in destructive processes elsewhere. Microglia does produce elsewhere compound granular corpuscles in the brain, just as connective tissue cells elsewhere in the body also become phagocytic. The specific stain for microglia is one of the best methods to demonstrate these macrophages elsewhere in the body. As to their derivation elsewhere, I am not qualified to discuss that, but it seems evident that they are mesodermal, and it is possible to put phagocytosis in the brain and elsewhere on the same footing. It adds unnecessary confusion to go back to the view that oligodendroglia and astrocytes can form these macrophages unless there is clear morphologic evidence of it with specific stains.

CHICAGO NEUROLOGICAL SOCIETY

Annual Meeting, May 7, 1927

DR. JOHN FAVILL, *President, in the Chair*

Demonstraton of Clinical Cases:

CHRONIC EPIDEMIC ENCEPHALITIS. DR. MEYER SOLOMON.

This patient, a single girl, aged 19, who was admitted to the neurologic department, Northwestern University Medical School, Dec. 29, 1926, has a family history and past history which are unimportant except that she had mild influenza at 12 and again at 13, and has had several attacks of tonsillitis in the past few years. Six months before the onset of the present illness she had what she thought was "a nervous breakdown," of unknown cause, characterized by crying and fatigue, without diplopia or lethargy, for which she was confined to bed for one week; no physician was called and she does not know positively whether she had fever, although she felt feverish for a couple of days. The onset of the present illness occurred two years ago, with nocturnal restlessness and insomnia which have continued to date. Six months later the tonsils were removed. Since then, for the past year and a half, the general nervous and mental complaints include: diurnal somnolence, so marked that she falls asleep repeatedly even while talking or working, which contrasts strongly with the nocturnal insomnia; nervousness, irritability, emotional instability, lack of pep and a constant tired feeling; a tendency to discouragement and crying, because of the inability to keep a job (she has held more than twenty jobs in the past two years), the symptoms and much quarreling and dissatisfaction at home due especially to the fact that she has been out of work. Onanism has occurred three or four times a week for a few years past. A sweetheart deserted her a few months ago. Discussion of the home situation leads to acute emotionalism with restlessness, crying, and panting breathing. The intellect is clear.

The somatic complaints are: The facial muscles frequently go into spasm so that she assumes peculiar forced facial expressions, lasting from one-half minute to one-half hour. While walking she has attacks of what she calls "tightening of the muscles" all over the body, especially of the arms and legs, so that often she must stop because she cannot move the upper or lower extremities; at other times she can continue walking; the attack lasts from a few minutes to a half hour, and is preceded or accompanied by an attack of tachypnea. The head is slightly bent forward, especially when walking; she has slight difficulty in getting started in walking, and says she drags herself along. The speech is nasal and she feels as if she has a cold in the head. Sensory symptoms are: paresthesias, pins and needles, especially of glove and stocking distribution over the hands and feet; bilateral tinnitus at night so that she frequently wakes up screaming from a terrifying dream and finds herself in an attack of tachypnea; vision was blurred two years ago, without diplopia, but is now corrected by glasses.

Complaints relating to the vegetative functions include: difficulty in breathing, with irregularly occurring attacks of tachypnea, for one-half year past, lasting from half a minute to half an hour or more; moderate salivation day and night, with the mouth constantly kept somewhat open; ravenous appetite, with sudden hunger attacks at night; marked sweating of the palms of the hands for the past year, and of the armpits for the past half year.

Examination gave negative results except as follows: head slightly bent forward and held stiffly, especially when walking; the arms swing normally; the thyroid is slightly and uniformly enlarged; tachypneic attacks, which were exaggerated by self-observation and cut short by distraction; recital and discussion of home conditions precipitated an attack of restlessness, panting

breathing and crying. The nose department reported the presence of some rhinopharyngitis and a deviated septum. The laboratory observations are: urine normal; blood Wassermann reaction negative; white blood cells 7,500, lymphocytes 51 per cent, large mononuclears 6, polymorphonuclear transitionals 41, polymorphonuclear basophils 2 per cent; red blood cells, 4,980,000; hemoglobin, 80 per cent; blood calcium normal (9.6 mg. per hundred cubic centimeters), blood sugar (glucose) approximately normal (83.3). Sugar tolerance is normal except that the blood sugar was lower (71.4) after one hour than at the beginning of the test (83.3); basal metabolic rate —15.4; the spinal fluid is clear, colorless, and contains one lymphocyte per cubic millimeter, Nonne test, phase I, is negative and phase II shows slight increase; the Wassermann and Lange tests are negative.

The diagnosis is chronic epidemic encephalitis, complicated by occupational and home maladjustments and conflicts. Superficially, there is much resemblance to an anxiety neurosis, hysteria and psychogenic tics.

The condition has greatly improved: sleep is fair; the dreams and screaming at night have ceased; there is still some tendency to fall asleep during the day and for the mouth to be open with some salivation; the sweating of the palms of the hands and of the armpits has ceased; the tachypneic attacks are less frequent and not so severe; she has more self-control and is more cheerful; the head is still slightly bent forward; she has more pep and is not so constantly tired. The facial spasms and hypertonic attacks in the extremities have ceased; the paresthesias no longer annoy her; the ravenous appetite has passed away. She and the family understand each other better. Occupational placement is now the main problem.

This case brings out clearly the importance of a minute history and careful examination for the diagnosis of chronic epidemic encephalitis of mild type, in the absence of a clear-cut, objective picture of parkinsonism. In the treatment of these patients it is important to distinguish between emotional upset due directly to the organic changes with superadded fatigue and intoxication, from the patient's concern over the organic disease, and from mental conflicts with maladjustment to other situations. The psychotherapeutic aspect, therefore, is not to be neglected.

THE MICROSCOPIC ANATOMY OF INFANTILE AMAUROTIC IDIOCY WITH SPECIAL REFERENCE TO THE EARLY CELL CHANGES AND THE INTRACELLULAR LIPOIDS. ROY R. GRINKER, M.D.

Two cases of amaurotic family idiocy of the infantile type were studied histologically and microchemically. The status spongiosus, described by Bielschowsky and Globus, was not noted nor were there seen the monstral glia cells found by Schaffer. A decrease of the granular layer of the cerebellum was found, not similar to the severe cerebellar condition in the cases of Bielschowsky and Greenfield and Holmes, as the glia was not proliferated. The process resembled a developmental defect more than an active process. In the thalamus normal cells were not seen; many were completely disintegrated, and neuroglia proliferation was present. Schaffer originally described marked loss of myelin substance in the thalamus, and Hassin recently localized the most severe changes in his case in that region. The loss of so many cerebral connections of the optic thalamus may mean that in this ganglion there is added to the primary pathologic condition, a secondary degeneration of the "Grosshirn Anteile."

Early in his studies, Schaffer utilized amaurotic idiocy for the study of the endocellular and pericellular nets, as he believed that the cell swelling made them more visible. He, Sachs and Strauss, and Vogt considered the intracellular net to be the neurofibrillar net described by Bethe as the intracellular conducting apparatus continuous with the peripheral neurofibrils. They compared the perinuclear dark ring to the perinuclear net of Donnagio, and

described a coarse pericellular or Golgi net. The dustlike granules were considered to be degenerated portions of the intracellular net, while the more resistant Golgi net only later degenerated. Economo and Bielschowsky showed, however, that the endocellular net is not of neurofibrillar origin, but is part of the cytoplasmic reticulum. They demonstrated that the Golgi net was only a condensation of the diffuse glia reticulum of Held about the cells, and that amaurotic idiocy was characterized by peripherally compressed neurofibrils. It was observed that the cytoplasmic reticulum, made visible by the abnormal lipoid material, ultimately degenerates. The neurofibrils which are pushed against the cell membrane later also disintegrate.

The gross abnormalities in the convolutions of the two brains studied have been found and described in detail by Schaffer from his material. He found disturbances in myelogenesis, pithecoid cortical layers and embryonal fissuring, and concluded that these changes were evidence of an embryonal weakness of the ectodermal structures which predisposed to the disease process. He considered that the essential pathologic condition was an initial swelling of the cell hyaloplasm with subsequent lipoidal degenerative changes. Bielschowsky, however, found intracellular lipoids without much swelling of the cells, and concluded that Schaffer's theory was faulty. He believed that the lipoidal change was a disturbance in the vegetative function of the cell. Mott believed that there was a deficiency of nucleoproteins and that the nucleus provided a splitting ferment but not a synthetic enzyme to convert the lipoids to simpler substances. Gordon and Savini considered an endocrine disturbance the basis of the pathologic change.

Hassin found well preserved Nissl bodies about the nucleus, as did Vogt in cells which had not reached their terminal stage, and believed the fundamental process to be an agenesis of Nissl substance, with secondary degenerative changes. This theory cannot be corroborated by this study.

The cases reported here revealed very early changes. The initial nuclear swelling and hyperchromatic appearance, the development of lipoid at the base of normally sized cells which contained their full quota of Nissl bodies and normal neurofibrils, and the gradual invasion of the cells by lipoids were the earliest pathologic abnormalities. Later the visible cytoplasmic reticulum degenerated, and still later the intracellular neurofibrils. The cell swelling was secondary and apparently was a result of distention by the lipoids. The fact that normal cells may be seen late in the course of the disease and that transition stages to the final disintegration of the cell can still be seen, gives rise to the belief that the brains, although they contain gross developmental defects, were composed of morphologically normal cells at birth.

Retinal Changes: The first studies of the retinal lesions by Mohr, Frank, and Kingdon and Russel gave the impression, because of the wide interganglionic spaces, that there was only an edema of the ganglionic internuclear layers. This appearance is now known to be a result in part of the loss of degenerated cells. Verhoeff, Hancock and Coats, and Cohen and Dixon have described the retinal changes in detail, which are in accord with the changes in the retinas described previously. The ganglion cells undergo the same process as the cortical cells. They become swollen and have a reticular cytoplasm containing lipoid, which stains bright blue with hematoxylin. Later, the cells disintegrate and are replaced by fibrous glia. In the infantile type no other retinal layer than the ganglionic and internuclear is affected, but as shown by Bielschowsky, Stock, and Greenfield and Holmes, in the juvenile type the outer nuclear layer is profoundly degenerated and is replaced by proliferated glia. The pigment layer is also definitely increased, invading the degenerated outer nuclear layer. On the other hand, cases in adults, which reveal in the brain the typical cell changes, may progress without retinal lesions, as was described by Schob and Walter. There does not seem to be any uniformity in the retinal pathologic condition in the various types of the disease, or any graded severity.

Lipoids: Wolfsohn and Oliver described an orange red material in scharlach preparations within the ganglion cells containing doubly refractile globules. In the glia and adventitial cells they found bright red droplets. Although they did not use differential methods, they concluded that the intracellular lipoid was a mixture of phosphatids and cholesterol esters which was gradually broken down from its complex form into simpler components by the glia and adventitial cells. Schaffer classified amaurotic idiocy according to the staining reactions of the intracellular substance. The infantile type contained prelipoid material which took a hematoxylin stain; the juvenile type stained well with scharlach R, and the adult type with osmic acid. He considered that all three types underwent the same process, the composition of the lipoid material becoming simpler with duration of the disease. Hurst, in his studies of the lipoids in various neuronic degenerations, concluded that the infantile and juvenile types varied only in their lipoid content. In his infantile cases there were readily soluble phosphatids and cerebrosides, while in his juvenile case the same lipoidal mixture did not take a hematoxylin stain and was difficultly soluble in all fat solvents. He thus found a gradual transition of the lipoid to the senile lipochrome pigment, which is insoluble in all reagents.

My study has yielded results similar to those of Hurst. The intracellular lipoid, with the exception of its resistance to complete solubility, reacted like normal myelin. Some readily soluble neutral fat was found in the gitter cells. A few doubly refractile crystals were found which were readily soluble in acetone. The remainder of the lipoid mass reacted like phosphatids and cerebrosides, but was never completely soluble in any reagent. Even in the process of celloidin embedding a bright golden pigment was still visible, as it was after hot xylol treatment. Hurst found the same atypical solubility and concluded that it was due to a lipoprotein combination.

If Schaffer's theory that the initial process is a swelling of the hyaloplasm resulting in the precipitation of the intracellular lipoids is correct, then, according to the physical property of emulsions, the entire lipoid material in the cell should precipitate out of its emulsion at once. However, the early cell changes which have been described demonstrated that the lipoid material appears first locally at the base of the cell and then gradually increases in amount. Little of the lipoid material is converted into a simpler fat; in only relatively few glia cells was neutral fat found. The resemblance of the lipoid in the glia to that in the ganglion cells does not necessarily mean that the glia has suffered the same type of degeneration and that the disease therefore affects the entire nervous ectoderm. The lipoid-containing glia cells are perineural in position and apparently have absorbed some of the ganglion cell contents. Their nuclei show signs of having reacted for the occasion.

Apparently, the lipoid is disposed of with difficulty, just as is the normal lipochrome pigment. In other types of neuronic degeneration Hurst finds that the phosphatid, cholesterol and cerebroside combination separates into its components and is readily converted into fatty acids and then quickly into neutral fat. In amaurotic idiocy, therefore, some strong combinations with the lipoids must exist which prevents their conversion into neutral fat. The atypical solubility, and the resemblance to the lipochrome pigment or "Abnutzungs Pigment," which is an accompaniment of senile changes within the cell, suggest a composition of the intracellular material in amaurotic idiocy, similar to that of senile lipochrome.

The gradual development in a brain with congenital defects of a slowly increasing amount of intracellular pigment resembling senile pigment makes it more than likely that the process is a premature senile change in the cell. This may be due to a faulty cellular metabolism, a disturbance in its vegetative functions, a generalized disturbance in lipoid metabolism, an endocrine disorder, or the presence of abnormal ferments. None of these can be demonstrated, and the microscopic picture may be identical. It is probable that primarily the cells are of normal morphology, and that after a variable period of apparently normal functioning they undergo a pathologic process similar

in its slowness and ultimate lipoid product to senility. Microscopic studies do not lead far toward a knowledge of the pathogenesis of amaurotic idiocy.

AMYOTROPHIC SYPHILITIC MENINGO-MYELITIS: REPORT OF TWO CASES WITH ARGYLL ROBERTSON PUPILS. THEODORE T. STONE.

Amyotrophic syphilitic meningomyelitis occurs not infrequently, and in many instances the causative factor is overlooked or not determined. Because of the fact that early and vigorous treatment in these cases may bring about a complete arrest and cure in many instances, it was considered important to study carefully two such cases that presented Argyll Robertson pupils in addition to muscular atrophy. The two cases were those of a woman, aged 48, and of a man, aged 56 years. Both of these patients developed atrophy of the small muscles of the hand with spastic paraplegia and a positive Wassermann reaction in the blood and spinal fluids. Typical Argyll Robertson pupils were present in each instance. The following conclusions seemed justified:

Syphilis is the most common cause of chronic spinal progressive muscular atrophy of the atonic type; i. e., the atrophy is spinal in origin, the paralysis is flaccid and the deep reflexes are changed, with no sensory disturbances. The reflexes are eliminated or abolished in the extremities in which wasting of muscles occurs, and there is no sensory loss in most cases. Bulbar symptoms apparently do not occur. There may be interference with sphincter control, either bladder or rectal. Early and vigorous antisyphilitic treatment may bring about an arrest of the wasting and weakness. The presence of Argyll Robertson pupils in amyotrophic meningomyelitis does not necessarily mean a poor prognosis.

PRIMARY GLIOMA OF THE CHIASM AND OPTIC NERVES ASSOCIATED WITH RECKLINGHAUSEN'S DISEASE. ALBERT B. YUDELSON.

DEMONSTRATION OF DECEREBRATE RIGIDITY. LEWIS J. POLLOCK and LOYAL DAVIS.

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

Regular Meeting, Thursday, May 19, 1927

HENRY VIETS, M.D., *President, in the Chair*

A CASE OF POSTENCEPHALITIC PARKINSONIAN DISEASE, WITH POLYDIPSIA. Presented by DR. J. W. COURTNEY.

In the ARCHIVES OF NEUROLOGY AND PSYCHIATRY **8**:40 (July) 1922, Kennedy, Davis and Hyslop reported, among others, a striking example of parkinsonism with polydipsia and polyuria. They preface their recital with the remark: "We believe that the significance of disturbed vegetative function in encephalitis can hardly be overstated; not only are such cases difficult to diagnose, but some of them may furnish valuable data for a future comprehension of neurotic and psychotic phenomena on a physical rather than on a purely emotional or psychic basis."

A similar case is that of a youth, aged 19, American born, whose family history is without nervous or mental stigmas. The patient was born at full term and without complications. There are three younger children in the family, all of whom are healthy. The patient had pertussis at 6 months, but no other illness in childhood; his development was normal. He went to school at 6 and was considered an unusually promising pupil.

At the age of 15, apparently without marked fever, the boy was observed to be drowsy constantly. He complained of incessant fatigue, and would lapse frequently into a sound sleep. Prior to this time he excelled in outdoor sports of all kinds. The contrast between his former activity and his present inertia

was striking; he barely had sufficient energy to dress himself, much less to leave the house. His interest in what went on about him diminished to the vanishing point. His speech was slow and slurring, and his bodily movements were as inelastic as those of an old man.

In the third year of the disorder the boy began to drink enormous quantities of water and to void a proportionate amount. Up to two months before examination the twenty-four-hour intake was from 12 to 13 quarts, and always during the daytime. During the month before presentation the consumption dropped to 8 or 9 quarts daily. The output of urine averaged from 10 to 12 quarts in the twenty-four hours. During the first three years the sclerae were constantly yellow. Drooling at night was incessant, and for the year before examination frequent yawning was annoying. To these were added episodes of extreme chilliness in which the teeth chattered and the gooseflesh appearance of the arms was particularly marked. There was no special respiratory trouble, and cyanosis was never observed.

Of peculiar interest was one symptom, generally regarded as purely psychic in origin, from which the patient suffered to a marked degree. The intellectual processes were not retarded; delusions, hallucinations and illusions were absent, and insight was intact; nevertheless, the youth was the victim of a compulsion; he could not pass the edge of a rug without kicking it up. As soon as this act was performed, he straightened the rug out again. He even tried to pry up the closely butted edges of an oilcloth floor covering with the toe of his boot. As he went up stairs he found himself compelled to kick the back-board of each tread. On the street he had to bring the toe of his boot sharply against every brick which stood above the general level of the sidewalk.

Physical examination revealed the following: The patient was 6 feet (183 cm.) tall and weighed 165 pounds (74.8 Kg.); his complexion was fair; the features were strikingly masklike, the body was inclined forward; the hands were carried in the customary parkinsonian manner and showed the characteristic pea-rolling tremor; the gait was somewhat festinating; retropulsion was present at times; the pupils and ocular muscles did not show any abnormalities; the knee jerks were present and fairly lively, the left somewhat more so than the right; the achilles jerks were normal; at times the right lower extremity showed a fairly coarse flexor-extensor tremor; the speech was somewhat staccato; the urine was particularly colorless and did not show any sediment on standing for twenty-four hours; its specific gravity was constantly 1.000; it was slightly acid and contained neither sugar nor albumin.

It has long been known that puncture of the medulla can produce polydipsia and polyuria. In epidemic encephalitis, a common port of entry for the pathogenic organism is through the nose and accessory sinuses; once the latter point is passed by this organism, the medulla lies not far distant in the path of least resistance.

I have long been convinced that, in the character, degree and extent of the remaining clinical manifestations of epidemic encephalitis, there are invaluable clues to the pathogenic mechanism of those phenomena, neurotic and psychotic so called, to which the paper by Kennedy, Hyslop and Davis refers. Even in fatal cases, the pathologic changes in the encephalon are not gross. Furthermore, many of the clinical syndromes produced by this disease are not different from those which at present are ineptly considered of purely psychogenic origin. I contend, therefore, that the psychologic interpretation of the syndromes in question should be dropped entirely, and that they should be regarded as due to pathologic changes in the encephalon, identical in degree, at least, with those which exist in certain stages of epidemic encephalitis.

DISCUSSION

DR. STANLEY COBB: Recently, there have been two such cases at the City Hospital. One was that of a youth, aged about 19, who for a year and a half had a left-sided parkinsonian syndrome with marked diabetes insipidus.

Pituitary extract stopped the polyuria, but all other symptoms increased; he had extreme headaches and increased rigidity. Apparently, the increase in the symptoms was caused by the pituitary extract. The second case was that of a patient who had a minor injury to the head about two years before admission; he attributed all his symptoms to that. About six months later he had influenza and diplopia. Then about six months later (one year before examination) he began to put on weight. In three or four months he gained almost 50 pounds (22.7 Kg.), and at admission he weighed about 240 pounds (108.8 Kg.). He should have weighed 180 or 190 pounds (81.6 or 86.2 Kg.). Neurologically he showed only a left extensor plantar reaction, and irregular, unequal pupils. With this history and these observations, it looks as if the case was one of encephalitis.

INDIVIDUAL PSYCHOLOGY AND THE TREATMENT OF NEUROSIS. Presented by DR. LEONHARD SIEF, Munich.

All human life, its preservation and its development are built on social intercourse. This fact has, however, two aspects, the one friendly, the other likely to be disturbing: of the aspects, namely, of cooperation and of competition, of fellow feeling and of will-to-power, which predominates in the individual?

"The child is father of the man." Every child will count, will be somebody. Starting from a wholly natural feeling of insecurity and inferiority (experienced in the comparison of himself with the stronger and more developed adults about him) he strives, following the law of balance, toward a goal of security and superiority, and molds his physical, psychic and mental creative powers in the service of this goal, in the interest of his desire to be "up" not "down." With reference to this goal, all his modes of expression get their "meaning" as responses to the social situation. Under the well oriented help of the educator he develops, little by little, his abilities and powers, self-confidence, courage, training, self-reliance and social relationships—and this even in case of organic inferiorities; while proporationately to such development, his feeling of inferiority and his will-to-power over others diminish. But if, on the other hand, he falls under the influence of an educator who is himself badly oriented and ill prepared for his work, then the inferiority feeling and the will-to-power become intensified at the cost of the social feelings. The result is pessimism, discouragement, egocentrism and all the tricks of a bad style of life, of maladjustment in greater or less degree: neurosis, perversions, delinquency, psychosis and suicide; for childhood is preparation, in the one direction for social life, in the other for neurosis and still more extreme aberrations.

Neurosis is a half-and-half adjustment to the tasks of life; it is a social disturbance; i. e., the neurotic person is cooperative so far as his superiority is approximately guaranteed. But in the moment when his superiority is endangered, in face of blame, defeat, decision or responsibility, he tends to secure his self-esteem with the "alibi" of nervous symptoms: fright, tiredness, sleeplessness, tears, tantrums, fighting, asthma, headache and so on: "What could I not accomplish if!—" It follows that the neurotic person is not always and in every direction neurotic; though his disposition, that is, his tendency to despondency, superiority and escape from real or seeming depreciation into the securing irresponsibility of neurosis, remains the same. Despite the common sense of such a person, neurosis is always a self-deception that he dares not "understand," because of the threatening feeling of insecurity.

The prognosis depends on the individual's dissatisfaction with his neurotic style of life, on his will to change and on the possibility of winning him out of his disheartenment into full cooperation. Neurosis is not inborn, but acquired; it is the result of a mistaken estimate of himself and of life into which the individual fell in childhood—an estimate not by any means forced on the child by the basic facts involved, but one none the less understandable.

There is not any obligation to neurosis, nor is it a real disease. And the last but not the least presupposition of healing is the rightly scientific and practical orientation of the physician. The individual of lesser development has need of one of greater development to help him overcome his lack.

Treatment: The first problem in treatment is that of creating an atmosphere of confidence and ease as the prerequisite of the second step—explanation and encouragement. In the first consultation the physician must be able to help the patient to talk freely about his complaints and his previous history, and to describe his present situation and former situations as far back as his earliest childhood (family, school, organ inferiorities, diseases, behavior, dreams, friendships and interests). Under what impressions and influences did he live and grow up? How did he react to them? What opinion did he form about himself, his ability and his chances in life? With whom has he, and had he earlier, the best relationship? With whom the worst? Childhood remembrances and recurring dreams are important as the characteristic expression of the whole social atmosphere and his response to it. They show best his characteristically securing attitude and his scheme of life, his guiding line—his inferiority feeling and its compensation, the goal of his superiority tendency. The purpose of the first interview must be to learn this behavior pattern in order to be able to foresee the patient's behavior under given circumstances, and to foresay—though the physician must be careful not to say at first more than the patient will be able to work up. One ought never to promise a cure, but to say only that recovery is possible and to throw the responsibility on the patient, so as to stir up his own endeavor and indispensable cooperation. The question "What shall I do?" could be answered thus: Go on doing as you have already done until you feel that you can venture to do it better! or, How can one learn gymnastics? Everything that could hurt the patient's self-esteem, all moralizing, sneering, insistence, menace and so on must be most carefully avoided.

The main task of the treatment is to win the patient out of his fright and isolation, to show him in a modest, friendly, encouraging way the "meaning," the "purpose," of all his different modes of expression, his neurotic symptoms included. His tendency to idealize in order to depreciate—also in relation to the physician—must be demonstrated to him as a consequence of his general attitude and as inconsistent with reality and cooperation.

The inferiority feeling is an understandable but not a justifiable error. The neurotic person did not succeed, because he did not trust himself and did not train in the right way (vicious circle). As to the fact of organ inferiority, it is not what is inherited that is decisive, but how one uses it. Besides, the goal of universal superiority is unrealizable, and the trouble not worth the cost. Little by little he has to be helped to find for himself and to understand the entire mechanism of his neurosis; he must be led to become suspicious of his neurotic attitudes in order that he may be maneuvered out of his isolation, his struggling attitude and his over-high expectations and won to self-reliance and cooperation as the best security. Great friendliness is required to help him to descend, step by step, from the throne of godlikeness and to become a simple, reasonable human being. Every progress has to be recognized as the result of his good training; and every relapse to be explained as the expression of his conceivable, but not necessary, fear about the next step forward. Briefly, the way to win his full cooperation is to arouse his self-confidence, his courage for a new training, his sense of reality and his feeling of responsibility; and, hand in hand with his conscious destruction of the will-to-power, to encourage the consciousness development of his fellow feelings in the direction of the common weal.

DISCUSSION

DR. PERCIVAL BAILEY: I shall not attempt to discuss Dr. Seif's presentation. I should be doing battle with borrowed weapons, not having had any experience with patients of this type. I should, however, like to protest against

explaining psychologic phenomena by means of sentiments. Dr. Seif and Dr. Adler explain much by means of the sentiment of inferiority (sentiment d'incomplétude de Janet). Such explanations seems to me incomplete, because sentiments are themselves secondary phenomena arising from disorders of conduct. Back of a sentiment one must search for the primary disorder of activity.

The same fundamental error underlies the recent criticism of the opinions of de Clérambault concerning the chronic systematized psychoses. Ceillier (*Encéphale*, April, 1927) founds his views of these psychoses on the sentiment of influence. But back of this sentiment one must seek for a more fundamental and primary phenomenon, and this is just what de Clérambault has done, whatever one may think of his Carlylian literary style and disjointed exposition.

PHILADELPHIA NEUROLOGICAL SOCIETY

Regular Meeting, May 27, 1927

M. A. Burns, M.D., *President, in the Chair*

Information to be Derived from Ventricular Studies Other than Ventriculography in the Study of Brain Tumors. Dr. Francis C. Grant.

No single factor is more vital to the successful extirpation of a tumor of the brain than precision in its localization. If the position of the neoplasm is accurately known, the operative maneuvers necessary for its removal may be carried out with a much greater chance of success. The methods of determining the region of the brain involved by the tumor are many and varied. A careful detailed history, with the chronology of the onset of the symptoms plus routine neurologic studies, retinoscopic examinations and visual perimetric tests, together with vestibular studies and roentgenoscopic films will furnish clear evidence of the site of the neoplasm in the majority of instances. In a certain number of cases however, although the presence of a tumor is unquestioned, no definite clue can be obtained as to its situation. Hitherto, to solve this problem of an unlocalized tumor of the brain, a ventriculogram was necessary. As experience with ventriculography increased, the effect on the size, shape and position of the ventricles by tumors in different areas of the brain has become more clearly appreciated. It soon became apparent that if a definite routine was adopted in tapping the lateral ventricles, estimating the amount of fluid removed from each and determining by the injection of a dye whether the interventricular foramina were patulous, a shrewd guess could be made as to the abnormality in the ventricular outline which the roentgenograms would subsequently reveal. This procedure of attempting to determine the position of the lateral ventricles by plunging for them from a fixed point, of estimating their relative size by the amount of fluid recoverable from each, and lastly of discovering whether or not the interventricular foramina are patulous by the injection of a dye has been termed by Dandy "ventricular estimation." It is a logical outgrowth of his brilliant conception of cerebral pneumography. As bilateral ventricular tap without the replacement of the fluid by air is unquestionably safer than ventriculography, this has seemed sufficient justification to attempt to discover the value and the limitations of this less hazardous method for the localization of tumors.

Four conditions must be met in order to draw accurate conclusions from ventricular estimation. It is necessary to appreciate clearly the effects on the ventricular system of a tumor in different regions of the brain. The operator should be thoroughly familiar with the normal position, size and depth from the surface of the lateral ventricles so that he can be certain that failure to reach either of them is due to an abnormality in their outline and not to an

error in his technic. The patient must cooperate sufficiently to permit a complete neurologic study, so that by obtaining normal responses from the different areas of the brain it can be determined which of them, because of unimpaired function, are obviously not involved by the tumor. The possibilities of error inherent in this method must be grasped, so that, if necessary, air may be introduced to outline the ventricles accurately if the evidence from an estimation alone seems inconclusive.

Broadly considered, tumors of the brain produce two types of changes in the ventricular system — symmetrical or asymmetrical variations in the size, shape and position of the lateral, third and fourth ventricles. Symmetrical dilation of the lateral ventricles is always due to internal hydrocephalus set up by a tumor obstructing the free circulation of the cerebrospinal fluid. To produce such an effect the tumor must be situated either in, or impinging on, the third ventricle, supratentorially or in the posterior fossa. Under these circumstances ventricular estimation would reveal the lateral ventricles in normal position, or nearer the cortex than usual. Both ventricles would show an increase in fluid content to approximately the same degree. Dandy states that it is necessary to obtain at least 30 cc. of fluid by free flow or aspiration from each ventricle to be safe in inferring that hydrocephalus exists.

It is particularly in cases of hydrocephalus that injection of a dye — indigo carmine — into one lateral ventricle may give important information. If, under these circumstances, this indicator is injected into one side and cannot be recovered from the other ventricle, it is good evidence that the interventricular foramens are obstructed, and that the tumor lies in this region.

Asymmetrical variations in the ventricular outline are always due to tumors lying in either cerebral hemisphere causing a shift in the position of one or both lateral ventricles with filling defects in the outline or even complete obliteration of one ventricle. Such changes are due to direct pressure from the neoplasm and not to secondary effects from interference with cerebrospinal fluid circulation. Under such conditions, it may be impossible to introduce the exploring cannula into one or the other lateral ventricle. If both can be tapped, the ventricle on one side contains much more fluid than its fellow — definite evidence of the inequality in size.

The ventricles should always be tapped from the same position. It is only through the development of an accurate technic in introducing the cannula that positive evidence of changes in position may be determined. Bilateral trephine openings are made 7 cm. above the occipital protuberance and 1.5 cm. lateral to the midline to avoid the longitudinal sinus. The cannula is directed to enter the vestibule of the lateral ventricle, its widest point. The occipitoparietal approach is used because the cannula passes through a relatively silent cerebral area, well behind the sensory cortex and above the visual tracts. From this position the ventricles should be encountered at a depth of from 4 to 5 cm., the cannula being directed slightly outward and in a horizontal plane, level with the tip of the ear. A shift in the position of a ventricle may be demonstrated by variations from these normal planes necessary to enter it with the cannula.

A complete neurologic examination, performed on a cooperative patient, is necessary to obtain the most accurate results from ventricular estimation. The negative evidence of unimpaired functions from various cortical areas, demonstrating that they are probably not involved by the tumor, must always be considered. For example, if it can be shown definitely that one lateral ventricle is much smaller than the other, obviously the tumor cannot be cerebellar or in the third ventricle because a symmetrical hydrocephalus does not exist. The neoplasm must lie in that cerebral hemisphere harboring the smaller ventricle. If all the active cortical areas of that hemisphere function normally, the tumor must be located in a silent area, for example, the frontal lobe or the right temporal lobe.

Furthermore, ventricular estimation, interpreted in the light of positive clinical observations will go far toward enabling the surgeon to place the

tumor accurately. The difficulty in differentiating clinically between frontal and cerebellar tumors is a neurologic tradition. If, on tapping, no hydrocephalus can be demonstrated and the ventricles are shown to be undilated and unequal, the lesion almost certainly lies above the tentorium, while if they can be shown to be equally dilated, the tumor is probably cerebellar.

In order to evaluate properly the usefulness of any test, its limitations must be clearly understood. Ventricular estimation may easily lead the surgeon into error unless certain facts are taken into consideration. If ventricular asymmetry can be clearly demonstrated, this is certain evidence of a cerebral hemispheric tumor situated on the side of the smaller ventricle. If the ventricles are undilated, equal in size, and normal in position the lesion is probably not cerebral. It may be a cerebellar tumor which has not obstructed the cerebrospinal fluid circulation sufficiently to cause hydrocephalus. At times the posterior horns may be absent as has been pointed out by Penfield. This may be deceptive, for the ventricle apparently lies deep in the hemisphere and may seem undilated even though a moderate hydrocephalus exists. I know of a case in which a cerebellar tumor was ruled out because absence of the posterior horns led the operator to believe that no hydrocephalus existed. A ventriculogram later corrected the error and demonstrated a fair degree of bilteral hydrocephalus with no posterior horns. An angle tumor was later successfully removed. Hydrocephalus means either a third ventricle tumor, behind the foramen of Monro, if the free passage of dye from one ventricle to the other proves these foramens patulous, or a cerebellar tumor. But a ventriculogram is necessary to make this differential localization unless the clinical evidence of cerebellar involvement is unequivocal. Occasionally a frontal lobe tumor will cause hydrocephalus by blocking the interventricular foramens. The dye test should be of value in these cases. As a rule, if hydrocephalus is present it is wiser to inject air and rely on an interpretation of the shadows seen on the roentgenograms.

In any intracranial procedure, release of intradural tension by ventricular tap will reduce hemorrhage and make a proper approach to the lesion possible, thus greatly facilitating its extirpation. It is always a wise precaution, therefore, to know something of the size of the ventricles before attacking any tumor, no matter how positively its position may be known. Furthermore, if, in a suspected cerebellar lesion, a tap for relief from pressure fails to demonstrate hydrocephalus, the operator must be certain of his ground on clinical or other evidence before he explores the posterior fossa. In the presence of increased pressure, cerebellar tumors always cause hydrocephalus.

In a series of twenty-six cases, in which ventricular estimation was employed as an aid in diagnosis, nineteen were subsequently verified at operation or autopsy. The interpretations made of the observations were correct in eighteen instances and incorrect in one. One patient died who was already stuporous from high intracranial pressure due to a large infiltrating glioma.

Conclusions.—(1) Ventricular estimation is a safer procedure than ventriculography. In experienced hands, with proper interpretation, it may often give information of equal value. (2) Asymmetry of the lateral ventricles is strong evidence of a cerebral tumor on the side of the smaller ventricle. (3) A symmetrical hydrocephalus usually indicates a cerebellar tumor. It is under these circumstances, however, that an error may be made from ventricular estimation alone unless the clinical syndrome points to the cerebellum. If doubt exists, it is wiser to make a ventriculogram in the presence of hydrocephalus.

ENCEPHALITIS. DR. FREDERIC H. LEAVITT.

Virginia C., not quite 12 years of age, was presented because of the rather unusual sequence of symptoms, presumably following an acute encephalitis, although there was no history of the usual symptoms associated with an acute condition. The history, as given by the mother, was that she was normal

prior to about two years before presentation, when she was struck on the left side of the head by a tin can filled with earth. She was not hurt badly, but a little blood ran down on her face and she became frightened. Within a few days after being struck, she developed "sniffles and snorts," that is, there was a ticlike movement in which she would pull her nose to one side and snort. That was the first unusual condition noticed. About the time that these tic movements were lessening in severity, the child became somnolent. The teacher in school reported that she would fall asleep for several minutes at a time and at home she would fall asleep while eating meals; this kept up day and night for several months. She was taken to a physician, who told the mother that this was due to a disease of the nose and tonsils; treatment was administered, but the condition was not altered. About the time of the termination of the somnolence, which lasted several months, the child developed a condition of extreme tachypnea. She would breathe very rapidly and have difficulty in catching her breath; in association with this, she would become cyanosed. The attacks were associated with a feeling of fear, and when she was brought to the clinic at the Childrens' Hospital, she presented the picture seen in the so-called "shell-shocked" veterans. I was inclined to believe, at that time, that it was a cardiovascular asthenia. Following the tachypnea, she had great acceleration of cardiac action. I counted from 140 to 160 beats to the minute, and frequently the heart beat so rapidly that it could not be counted. She then became cyanotic again, short of breath, and very apprehensive. These symptoms subsided as the others had, and about three weeks ago she began to show a personality change. Instead of being neat and obedient she became the opposite. She used language such as the mother had never heard. She took things that did not belong to her from the house, and if any one took them away from her, she developed a temper tantrum. She became unruly and disobedient. The mother said that it was almost impossible to care for her at home; she was brought to the Orthopaedic Hospital but had to be removed because of her unrestrained actions; she would wander about, and attack the nurses.

This patient is of interest because of the various stages and changes in her condition: the somnolence, which disappeared; the tic movements, which disappeared; the tachypnea and the tachycardia, and the character changes. She also showed a picture of beginning parkinsonism: posture, station, facial expression (when not excited) and manner of speech.

DISCUSSION

DR. CHARLES W. BURR: Personality changes do not, in my experience, occur in patients who show marked parkinsonism. More and more cases of encephalitis without acute onset are appearing.

AMAUROTIC FAMILY IDIOCY. CLINICOPATHOLOGIC STUDY OF A CASE. DRS. MEYER SOLIS-COHEN, A. S. HADLER AND N. W. WINKELMAN.

A Jewish girl, aged 15 months, was admitted to the Jewish Hospital because of convulsions. There was a history of mental disease on both sides of the family. Another child had had a similar condition at the age of 17 months, and the condition was diagnosed hydrocephalus; postmortem studies were not permitted. The patient was a full term child and was breast fed for two months. At the third or fourth month it was noted that she was unusually sensitive to sounds, starting at the least noise. At 6 months the mother noted that the child's head was larger than normal, and at this time the child could not handle herself as well as a normal child. Convulsions began at about 3 months. On examination, the child appeared well developed, but her head measured 18½ inches (46.99 cm.) in circumference. There were convulsive movements with deviation of the head and eyes. The spinal fluid was under increased pressure (25 mm. of mercury). The eyegrounds showed the characteristic cherry-red spot. The extremities appeared somewhat flaccid, but the

reflexes were slightly increased. Little, if any, vision was present. Progressive emaciation occurred, with irregular fever and death ten weeks after admission.

At autopsy, the increase in the size of the head was noted. The brain was larger than usual and was as firm as if it had been in formaldehyde for weeks. The convolutional pattern was peculiar in that great variation in the size of the individual convolutions occurred, some being extremely small and others very large. On microscopic study, the most striking feature was the universal presence of the so-called Schaffer type of ganglion cell disease. Practically every cell in the central nervous system was swollen. Gitter cells occurred between the ganglion cells, and were especially marked in areas in which the pathologic process was most advanced. The Marchi stain demonstrated at a glance the enormous amount of fat within the cortex. Bielschowsky preparations gave characteristic pictures. The cerebellum was normal in general makeup, but the Purkinje cells likewise showed the typical cell changes. The granule cells were not altered. Demyelinated areas were present throughout. Glial proliferation could be demonstrated by special staining methods.

SOCIÉTÉ DE NEUROLOGIE DE PARIS

May 31, 1927

PROFESSOR G. ROUSSY, *in the Chair*

Rev. neurol. **1**:1036, 1927

TREATMENT OF GENERAL PARALYSIS WITH STOVARSOL. SÉZARY and BARBÉ.

Twenty-two patients were described, who were considerably improved following treatment with stovarsol. Sixteen had returned to their previous occupations; five still showed some symptoms, and one died of pneumonia. The duration of the condition in half of them was more than a year. In 20 per cent, benefit did not result. The serologic reactions were moderately benefited, although a complete recovery was not noted.

In the treatment, 21 Gm. was administered in a course of fifteen injections, and the course was repeated after a variable length of time.

Visual disturbances occurred in 7 per cent, and two patients went blind.

DYSBASIA LORDOTICA POSTENCEPHALITICA. ROGER, SIMÉON and DENIZET.

Following encephalitis, a youth, aged 20, presented a disturbance in gait consisting of exaggerated lordosis which practically threw him over backward with the trunk slightly rotated and inclined to the right. It disappeared when he ran and when he pushed a chair before him. When he walked upstairs, the attitude became more and more pronounced as he progressed. Slight parkinsonism was present. Torsion spasm or other hyperkinesia was not present. It would seem that in this case dystonia of the muscles of the dorsal plane, rather than insufficient tonus of the muscles of the ventral plane, was present.

INTRAMEDULLARY TUMOR, LOCALIZED BY IODIZED OIL: OPERATION AND RECOVERY. SICARD, HAGUENAU and WALLICH.

In the case reported, few clinical signs were present, in contrast with the marked physiologic disturbance, so that intramedullary tumor was diagnosed clinically.

The patient had pain in the lumbar region radiating into the lower limbs, without disorders of sensation or disturbed tendon reflexes. Slight contracture of the lumbar muscles was present, but disability of the sphincters was not noted. The presence of syphilis was ruled out, and lumbar puncture revealed

marked albumino-cytologic dissociation. Iodized oil 40 per cent was partially arrested at the level of the eleventh thoracic vertebra.

At operation, the posterior septum was opened and a typical glioma was enucleated.

Following operation, aside from disturbances of action of the sphincters, the course was benign until the ninth day, when complete paralysis of the lower limbs resulted from secondary hemorrhage. This, however, cleared up rapidly, and two months following the operation the patient was able to walk normally and was relieved of pain.

Spinal Dysesthesia Resembling Electric Shocks Produced by Flexion of the Head in Disseminated Sclerosis. H. Roger, J. Reboul-Lachaux and G. Aymes.

Four cases are reported, in which various movements of the head provoked painful sensations radiating down the limbs, which were strictly comparable to faradic electricity. They were also obtained by movements of the trunk and of the limbs. They greatly resembled the painful sensations described by Barré in cases of medullary compression, which he spoke of as cordonal in type.

The Pressure of the Cerebrospinal Fluid After Lumbar Puncture. Dr. O. Balduzzi.

Measurement of the intradural pressure was taken at varying periods following a preliminary lumbar puncture with removal of 5 and 15 cc. of fluid. Instead of the expected fall in pressure, there was a secondary hypertension which reached its limit in about six hours, was roughly proportional to the amount of fluid withdrawn and disappeared in twenty-four hours. This may furnish physicians with a more rational method of treating the complications of lumbar puncture.

In one case, the author followed the lumbar puncture with intravenous injections of glucose and witnessed the rapid disappearance of all disturbances.

Transitory Babinski Sign in a Case of Dementia Praecox. Henri Claude, Georges Bourguignon and Baruk.

The patient presented a typical catatonic condition, with negativism, and it was noted occasionally that the plantar reflex varied from flexion to extension in type. The course was followed with determinations of chronaxia, and it was disclosed that during the period that a positive Babinski sign was present the chronaxia of the extensor proprius was 0.68, whereas that of the flexor brevis was 0.29. When the sign changed to normal, the chronaxia of the extensor proprius was 0.24 and that of the flexor brevis rose to 0.44.

"Thus in this case the chronaxia corroborated the existence of a Babinski sign and explained its transitory appearance. This fact allows us to demonstrate objectively transitory modification of nervous functions in dementia praecox, which disclosed themselves in this case by the occurrence of the fleeting pyramidal disorder."

Book Reviews

PSYCHOKATHARTISCHE BEHANDLUNG NERVÖSER STÖRUNGEN. By DR. LUDWIG FRANK. Price, 10 marks. Pp. 208. Leipsig: George Thieme, 1927.

In science, as in commerce, when an innovation succeeds or attracts marked attention a procession of imitations soon follows in its wake. These may consist of some slight alteration in an unessential feature, minor improvement or merely the offering of the same article in a different, perhaps more attractive, wrapper. The advocate of the spurious product may unconsciously reveal the source of his inspiration by averring that it is "just as good" as the original. This brings us to the topic of psychocatharsis which, as the reader may surmise, is a near relative of the better known process of analysis. Those familiar with the development of psychoanalysis know that psychocatharsis with the patient in a state of hypnosis was a forerunner of the present procedure, and that catharsis, consisting in the release of repressed psychic affects, is still a vital part, though by no means the whole, of psychoanalytic procedure. There are good reasons why catharsis per se has been found to be ineffectual, in most cases, in permanently relieving neurotic symptoms, but Frank in a reactionary mood, reiterates that the essential factor in disease conditions embraced under the term "neurosis," which for some inexplicable reason he prefers to call "thymopathy," is an accumulation of affect stimulators. He defines affect stimulators as "those dynamic forces which instigate the stimulation of various affects" and attempts to summarize the differences between his cathartic treatment and freudian "dream analysis." Frank naively asks if "it is not simpler and more pertinent to allay the subconscious accumulated affect dynamics and to enlighten the patient in regard to sexual processes" which, he claims, is "the shortest road to the cure of so-called neurasthenic states."

Psychocatharsis is performed while the patient is in a state of half sleep, which is about identical with the passive position which the patient assumes on the couch in ordinary psychoanalytic procedure. Frank claims extraordinary results from this psychocathartic treatment even to the extent that "all patients suffering from angioneuroses" were essentially benefited and even cured (p. 144); likewise, all traumatic neuroses (p. 161) all sexual anomalies (p. 127), etc. To be sure, with psychocatharsis he combines occupational therapy, change of environment and persuasion; he stresses also the elasticity of his activity and the essential intuitive gifts and ingenuity of the physician.

As one reads Frank's book one becomes more and more impressed with the conclusion that his acquaintance with the development of psychoanalysis during the past fifteen years is fragmentary, and that he has failed to grasp the valid reasons why catharsis and confession alone have been unsuccessful at the hands of the men to whom he acknowledges psychotherapy is most indebted for its present status. As Frank does not delve profoundly into the intricacies of the unconscious, does not worry greatly as to the nature or genesis of anxiety, does not examine critically the adequacy of his explanations of the symptoms, this book will appeal only to those who are content with one extremely simple conception of a fundamental but isolated force in the general structure and therapy of psychoanalysis.

CONVULSIONS ET ÉPILEPSIE DES ENFANTS. By ANDRÉ COLLIN. Pp. 200. Price, 20 francs. Paris: Gaston Doin & Cie., 1926.

In this volume, the late author gives his experiences in a long neuropediatric practice. Trained as both clinician and neurophysiologist, he bases his views not only on personal clinical experience but also on a broad knowledge of fundamentals. Convulsions are common in children during the first years of

life (période précortico-active). These convulsions are usually benign in nature and the prognosis is favorable, the task of the pediatrician being to recognize in these first infantile convulsions those which can degenerate into true epilepsy. The criteria for benignity of infantile convulsions are: (1) they occur during hyperthemia; (2) they are usually generalized, with prominence of clonic contraction; (3) the bluish discoloration of the face is not so pronounced; (4) the stertorous period is not observed; (5) they are not followed by the cloudy state of sleepiness and apathy. On the other hand, apyretic convulsions beginning on one side, strictly tonic and followed by the stertorous phase and with an apathetic after-period, carry with them a guarded prognosis, as they are likely to become epileptic.

The author thinks that the mechanism of the benign and epileptic infantile convulsions is fundamentally different. The former may occur because the cortex is not yet wholly developed and therefore the volume capacity for nerve tissue energy is reduced; this leaves the subcortical centers always on the verge of overflow and discharge by convulsion on the occasion of intoxication, fever, digestive trouble or merely strong peripheral stimulation. In true epileptic convulsions, the nervous discharge is induced from the cortex. The cortex is an active agent of convulsions — by excitation or irritation.

The author gives much attention to methods of treatment in accordance with a firmly established tradition of the French school that the rôle played by heredosyphilis is important in infantile convulsions in general, as well as in those which are forerunners of epilepsy. The book has no adequate index, and references to other investigators are few.

BIRTH INJURIES OF THE CENTRAL NERVOUS SYSTEM. Part I. Cerebral Birth Injuries, by FRANK R. FORD. Part II. Cord Birth Injuries, by BRONSON CROTHERS and MARIAN C. PUTNAM. Pp. 164. Price, $4.00. Baltimore: Williams and Wilkins, 1927.

The book is divided into two parts. The first is on cerebral injuries at birth and their results, by F. R. Ford, and the second part on obstetric injuries to the spinal cord, by Bronson Crothers and Marian C. Putnam.

In about seventy pages, Ford discusses adequately the subject of cerebral injuries at birth, which always has been of utmost interest to neurologists. He comes to interesting conclusions: That there is convincing evidence that congenital diplegias, which constitute by far the largest group of the infantile spastic palsies, are the result of pathologic processes of intra-uterine origin and that they have usually been thought to be the result of injuries at birth. He does not come to any final conclusion regarding the causes of chronic hydrocephalus. Most of them are undoubtedly due to trauma at birth. The real injuries to the brain at birth, according to Ford, are caused by intracerebral hemorrhage and necrosis, by depressed fractures with laceration of the brain, and by more or less incapsulated meningeal tumors which compress and soften the cortex. In the material at the author's disposal, only 17 of 180 infantile cerebral palsies, or about 6 per cent, were due to injury at birth. Only from 2 to 3 per cent of epilepsies are due to injury at birth, and severe grades of mental defect are probably not related to injury at birth.

The second part relating to injuries of the cord at birth is equally interesting. Bronson and Putnam call attention to the frequency of injuries of the spinal cord due to the application of traction during delivery and comment on the striking fact that no emphasis is placed on this subject by most authors of obstetric textbooks. They are unable to state the exact percentage of such cases, but they consider the situation serious. They present twenty-eight cases. The manner of presentation is excellent. In practically all of them there is a photograph of the patient on one side, with a diagrammatic representation of the nature and location of the lesion with the symptoms.

Altogether, this is an excellent bit of work and the authors are to be congratulated.

THE INFLUENCE OF MUSIC ON BEHAVIOR. By CHARLES M. DISERENS. Price, $2.50. Pp. 210. Princeton, N. J.: Princeton University Press, 1926.

Most essays presented as part of the requirements for the degree of Doctor of Philosophy are not of great merit, but this thesis on the influence of music on behavior is an exception, for the author has presented a readable, interesting and informative book on a subject which today is arousing considerable interest. The author agrees with Darwin that music plays an important rôle in sexual selection, particularly in birds and insects. In mammals, the evidence is more doubtful but it seems to have operated in the case of anthropoids and simians, "where the male vocal organs are larger and the voice may be a charm to the female." The introduction, in fact, gives a good historical survey of the views of Darwin, Spencer and others regarding the development of music.

The book itself is divided into nine chapters with the following headings: (1) Introduction; (2) The Reactions of Animals to Music; (3) Music in Myth; (4) Music in Magic; (5) The Influence of Music on the Sick; (6) The Influence of Music on Work; (7) The History of Experimental Work on Reactions to Musical Stimuli; (8) An Experimental Study of the Influence of Music on Behavior; (9) General Summary and Conclusions.

In chapter 8, the author gives an account of an experimental study of the influence of music on behavior in the ordinary activities of daily life. His general conclusions are that music tends to reduce or delay fatigue and consequently increases muscular endurance, speeds up voluntary activity and influences the electrical conductivity of the human body as manifested in the psychogalvanic reflex.

EDUARD R. HOFMANNS LEHRBUCH DER GERICHTLICHEN MEDIZIN MIT GLEICHMÄSSIGER BERÜCKSICHTIGUNG DER DEUTSCHEN UND ÖSTERREICHISCHEN BESETZGEBUNG. By DR. ALVIN HABERDA; with a complete rewriting of the psychiatric section by DR. JULIUS WAGNER-JAUREGG. Ed. 11. Price, paper, 57 marks; bound, 61.80 marks. Pp. 1234, with 333 illustrations. Berlin and Vienna: Urban and Schwarzenberg, 1927.

Hofmann's textbook on forensic medicine, which appeared first in 1878, is too well and favorably known to need much introduction. It is a monumental work of reference, admirably illustrated and documented, which should be available to every physician who is interested in legal medicine. This new edition, the eleventh, has been thoroughly revised and brought up to date and many new illustrations have been added. A feature of particular interest to neurologists and psychiatrists is the newly prepared section on the legal aspects of mental diseases and defects, which has been prepared by Wagner-Jauregg in his usual clear and lucid style; it is fully up to the high standards set by Hofmann and Haberda for the rest of the book. This phase of the subject, which occupies about 120 pages, is considered under the heads of: (1) responsibility for crime; (2) business capacity; (3) evidentiary capacity; (4) detention under commitment and (5) marriage and divorce. The author never loses sight of the strictly medical functions of the expert and his discussions of the German and Austrian laws contain much that will be of interest to the American reader.

UEBER HYSTERIE. By ERNST KRETSCHMER. Second edition. Price, 6 marks. Pp. 128. Leipzig: Georg Thieme, 1927.

This volume has been translated into English by Oswald H. Boltz and was reviewed in the ARCHIVES OF NEUROLOGY AND PSYCHIATRY **16**:677 (Nov.) 1926.